STUDY ON ETIOLOGICAL PROFILE OF NEW ONSET SEIZURES IN ADULTS IN A TERTIARY CARE CENTRE

A DISSERTATION SUBMITTED TO

THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

BY

Dr. SUSAN GEORGE

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE AWARD OF THE DEGREE OF

DOCTOR OF MEDICINE - BRANCH I

(GENERAL MEDICINE)

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI – 11, TAMIL NADU

MAY 2019

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled "**STUDY ON ETIOLOGICAL PROFILE OF NEW ONSET SEIZURES IN ADULTS IN A TERTIARY CARE CENTRE**" submitted by **Dr. SUSAN GEORGE** to The Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfilment for the award of M.D Degree (GENERAL MEDICINE) is a bonafide work carried out by her under my guidance and supervision during the course of study from **2016** to **2019**.

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DECLARATION

I solemnly declare that the dissertation titled **"STUDY ON ETIOLOGICAL PROFILE OF NEW ONSET SEIZURES IN ADULTS IN A TERTIARY CARE CENTRE"** is prepared by me under guidance of **Prof. Dr. L. RAJAGOPALA MARTHANDAM, M. D**. The dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirements for the award of M.D Degree (Branch I) in General Medicine. I also declare that this bonafide work or a part of this work was not submitted by me or others for any award, degree, diploma to any university, found either in India or abroad.

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DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE STUDENT
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Dear Dr.SUSAN GEORGE, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting Held on 10.03.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of The Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

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1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of The validity
4. An annual status report should be submitted.
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This is to certify that I have verified this dissertation work titled "**STUDY ON ETIOLOGICAL PROFILE OF NEW ONSET SEIZURES IN ADULTS IN A TERTIARY CARE CENTRE"** of the candidate **Dr.SUSAN GEORGE** with registration Number **201611359** for the award of M.D. in the branch of General Medicine. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **3 percentage** of plagiarism in the dissertation.

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INTRODUCTION

Seizures are one of the most common neurological disorders we encounter in our day-to-day medical practice. Term ‘seizures’ dates back to 6th century, it is a word of Latin origin which means “to take possession of”, whereas the term epilepsy is a Greek word which means "to be seized by forces from without". Electrical changes occurring in brain manifest as seizures and usually are suggestive of underlying neurological diseases. Myriad are the causes that can precipitate a seizure, most of which were unknown until the advent of EEG, CT and MRI scans in the twentieth century. Thereafter, multitudes of studies have been conducted to decipher the causes, the most common among the causes were idiopathic, alcohol withdrawal, cerebrovascular accident, and CNS infections. These causes vary between countries and states depending on geographic location, socio-economic status, demographic features and genetics.

The most frequent risk factors for seizures in the elderly are cerebrovascular accidents followed by tumours and metabolic disorders. Tuberculosis related causes are one of the most common causes of seizures in all age groups. Small, single CT lesions which were diagnosed as tuberculoma previously, more recently have been correctly diagnosed as neurocysticercosis. Similarly, cerebral venous thrombosis is common in postpartum women, in India, which usually causes focal seizures but rarely can worsen into life-threatening status epilepticus.

Khadilkar et al[1] based on their recent community-based surveys showed that the epidemiological indices of seizures in India are comparable to that of the developed world with a prevalence rate of ~5 per 1000, but there is an increased

prevalence of epilepsies caused by neurological infections, trauma and perinatal distress. Hence this study was undertaken to identify the most common causes of new onset seizures in South Tamil Nadu and also to enlist various rarer aetiologies which may present as seizures of new onset.

AIMS AND OBJECTIVES

- To know about various causes of new-onset seizures in adults.
- To find out the most common cause of new-onset seizures in various age groups in this region.

REVIEW OF LITERATURE

HISTORY

The earliest descriptions of epileptic seizures have been recorded in the ancient Akkadian texts[2] from Mesopotamia, 2000 B.C. It was diagnosed as the 'hand of sin' (antasubbu) by the exorcists, caused due to the Moon God. The Edwin Smith papyrus dating to 1700 B.C also mentions epilepsy. Old Babylonian medical text 'Sakikku' went a notch ahead in detailing the types seen today including simple, complex seizures and narcolepsy.

In the Indian context, Atreya was one among the first to attribute seizure to brain dysfunction rather than divine intervention. Caraka Samhita Sutra dating to 600 B.C defined and classified seizures (Abasmara).

'On the sacred disease', a classic treatise authored by the father of medicine, Hippocrates, is accredited with the first formal description of this condition. He named it the 'Great Disease' from where the term 'grand mal' arose. 'Epilepsy', as a word is a derivative of Greek 'Epilepsia', meaning 'to take hold of'[3].

In the 19th century, French and English medicine, including eminent physicians like Brown Sequard, Robert Todd, Astley Cooper gave extensive accounts of the condition. It was in this time period that neurology became a new discipline, shunning the stigma around seizures. However, it was the pathological and anatomical studies of John Hughlings Jackson, father of modern epileptology,

helped give a new and accurate explanation of the condition. Similarly, Bromide introduced by Sir Charles Locock[3] became the first effective anti-epileptic.

John Hughlings Jackson, Father of modern epileptology

Hans Berger, a German neurologist, recorded the very first human electroencephalogram in 1925 and reported his findings five years later[3]. This documented the presence of electric discharges in brain and changes in the pattern of brainwaves in association with different seizure types. EEG helped in localizing the seat of the lesion which broadened the horizon of treatment options by incorporating surgical methods also. These included hemispherectomy by Dandy, callosotomy by Wagenen and Herren, temporal lobectomy by Bailey etc[4].

The next development was in the field of imaging like computer tomography (CT) scanning, magnetic resonance imaging (MRI), positron emission tomography (PET) which gave both structural and functional details of the brain.

Phenobarbitone and phenytoin were the ‘go to’ drugs for epilepsy in the early twentieth century. Improvements in the understanding of the electrophysiology of brain and seizures led to the discovery of several other drugs affecting the excitatory and inhibitory neurotransmitters of the brain.

Despite these advancements in the diagnosis and management of seizures, a greater chunk of the population in developing countries with this disorder are still undertreated because of the older supernatural views, discrimination, social stigma and poor access. To help in this regard, The International League Against Epilepsy was founded in 1909. Another equivalent organization is The International Bureau for Epilepsy, both of which have joined hands with WHO in 1997 to create public awareness.

EPIDEMIOLOGY

As one of the most common neurological condition, about 8- 10 % of the population develop seizures at one point in their lifetime, but only 2-3 % will progress to develop epilepsy[5]. In India, the incidence of epilepsy is 50 per 100000 and a prevalence of 5.59 per 1000 according to some community-based surveys[6]. The definition of epilepsy has been changing with time. The earlier definition of 2 unprovoked seizures separated by 24 hours has been replaced by a new one defined by ILAE. It defines the condition as one unprovoked seizure and a probability of further seizures similar to the general recurrence risk after two unprovoked seizures (at least60%) occurring over the next 10 years[7].

In a systematic review of epidemiology of seizures, Banerjee et al[8] found that the incidence and prevalence of epilepsy were lower in developed countries in North America and Europe compared to higher rates in Latin America and Africa. However, the Asian region reported the lowest frequency probably reflecting the cultural stigmatization. Demographic factors like age, gender, race and socio-economic factors have a take on the general incidence and prevalence of epilepsy. The incidence is high in infancy and early childhood in both developed and underdeveloped regions. But in underdeveloped regions, the next peak is in adulthood unlike that in the elderly population of the developed regions. The differences by gender and race have been reported in a few studies but are not statistically significant.

In a similar study in India[9], which it ought to contribute to one-sixth of the global burden, a bimodal distribution of epilepsy with the first peak in infancy and early childhood followed by a second peak in elderly have been reported, both with varying aetiologies. Socioeconomic factors like education, occupation, and income are closely associated with factors like birth trauma, infection, poor nutrition, poor hygiene, and poor health-seeking behaviour, which in turn influence the risk of epilepsy and its prognosis. This was also reflected in the wide divide seen between the urban and rural population, highlighting the need for increased service in this region.

DEFINITION

A seizure has been defined[7] as ‘a transient occurrence of signs and/or symptoms due to an abnormal excessive or synchronous neuronal activity in the

brain'. It is a paroxysmal event of excessive neuronal discharge from the central nervous system. The variability in the clinical manifestations of the condition ranging from generalized convulsions to that is not discernable by bystanders is due to the differences in the origin of this excess activity and its spread over the brain surface.

They were usually described using terms like grand mal and petit mal which were imprecise. The next step in classification was dividing it into partial and generalized. The term partial was intended for seizures starting at one area or side of the brain. Generalized seizures involved both the cerebral hemispheres at the same time. Partial seizures were further classified into simple and complex seizures based on whether the patient was conscious or not. In the former, the patient is aware of the event whereas in complex seizures a patient has impaired awareness. The drawback of this older classification was that it couldn't account for all the types. Hence there was a need to reclassify seizures.

ILAE has been instrumental in defining and classifying seizures which laid the foundation for the understanding of the many types that couldn't be included in the older classifications. Over the time they developed 2 classifications: Clinical and Electroencephalographic classification of Epileptic Seizures in 1981 and International Classification of Epilepsies and Epileptic Syndromes in 1989. They further revised their classification in 2017 to make the diagnosis and understanding easier[10].

CLASSIFICATION

The new basic classification (2017) takes into account 3 key features namely

1. Where seizures began
2. Level of awareness
3. Other features

DESCRIBING ONSET

This first point of identifying where seizures began has its importance in that it affects the type of medication, need for surgery, probable cause and thus prognosticates the condition. Based on this fact alone 4 types of seizures can be identified

- **Focal seizures:** are the (older) partial seizures, which have a focus of origin localized to one side of the brain
- **Generalized seizures:** are the primary generalized seizures which are engaging the neuronal network on both the sides from onset itself.
- **Unknown onset:** where the onset of a seizure is not known. This can be reassigned later on if it becomes known.
- **Focal to bilateral seizures:** are the secondary generalized seizures which begin as focal seizures. The term generalized is now reserved for the ones at the start of a seizure.

DESCRIBING AWARENESS

Awareness of a patient has more of practical importance concerned with the safety of the patient and is different from the consciousness which might be difficult to evaluate.

- **Focal aware:** when the awareness is intact without considering whether the patient was able to communicate or respond during the event.
- **Focal impaired awareness:** awareness is impaired at some point during the episode despite having a vague idea of the event. This term replaces complex partial seizures.
- **Awareness unknown:** when the status can't be ascertained as in the case of no witness or a night episode.
- **Generalized seizures:** are presumed to affect a person's consciousness or awareness without requiring any special terms for the same.

DESCRIBING OTHER FEATURES

In the new basic classification, seizure behaviours are segregated based on whether they involve movement.

- **Focal motor seizure:** which means some particular movement occurs during the seizure episode which can range from simple stiffening to twitching or jerking movements of any part of the body or automatisms (rubbing hands, licking lips, walking or running).
- **Focal non-motor seizure:** which has symptoms other than movements, like any change in emotions, sensations, experiences or thinking.

- **Auras:** depicts any symptom that the patient feels at the beginning of an episode. This term has been omitted from the new classification.

ILAE 2017 New Basic Classification[10]

1) FOCAL ONSET

- Aware/impaired awareness
- Motor onset/ non-motor onset
- Focal to bilateral tonic-clonic

2) GENERALIZED ONSET

- Motor
  - i- Tonic-clonic
  - ii- Other motor
- Non-motor (absence)

3) UNKNOWN ONSET

- Motor
  - i- Tonic-clonic
  - ii- Other motor
- Non-motor
- UNCLASSIFIED

DESCRIBING GENERALIZED ONSET SEIZURES

Those which arise from both the sides of the brain are called generalized onset seizures which are of two types.

- **Generalized motor seizures:** GTCS or generalized tonic-clonic seizure is still used which corresponds to ‘grand mal' seizures. The term refers to the description of the condition with stiffening (tonic) and jerking (clonic).
- **Generalized non-motor seizure:** this term primarily depicts absence seizures which correspond to ‘petit mal'.

ILAE 2017 New Expanded Classification

This adds further subheadings into the main framework of new basic classification.

1. FOCAL ONSET

- Aware/ impaired awareness
- Motor onset
    - i- Automatisms
    - ii- Atonic
    - iii- Clonic
    - iv- Epileptic spasms
    - v- Hyperkinetic
    - vi- Myoclonic
    - vii- Tonic
- Non-motor onset
    - i- Autonomic
    - ii- Behaviour arrest
    - iii- Cognitive

iv- Emotional

v- Sensory

- Focal to bilateral clonic

2. GENERALIZED ONSET

- Motor

i- Tonic-clonic

ii- Clonic

iii- Tonic

iv- Myoclonic

v- Myoclonic-tonic-clonic

vi- Myoclonic-atonic

vii- Atonic

viii- Epileptic spasms

- Non-motor (absence)

i- Typical

ii- Atypical

iii- Myoclonic

iv- Eyelid myoclonia

3. UNKNOWN ONSET

- Motor

i- Tonic-clonic

ii- Epileptic spasms

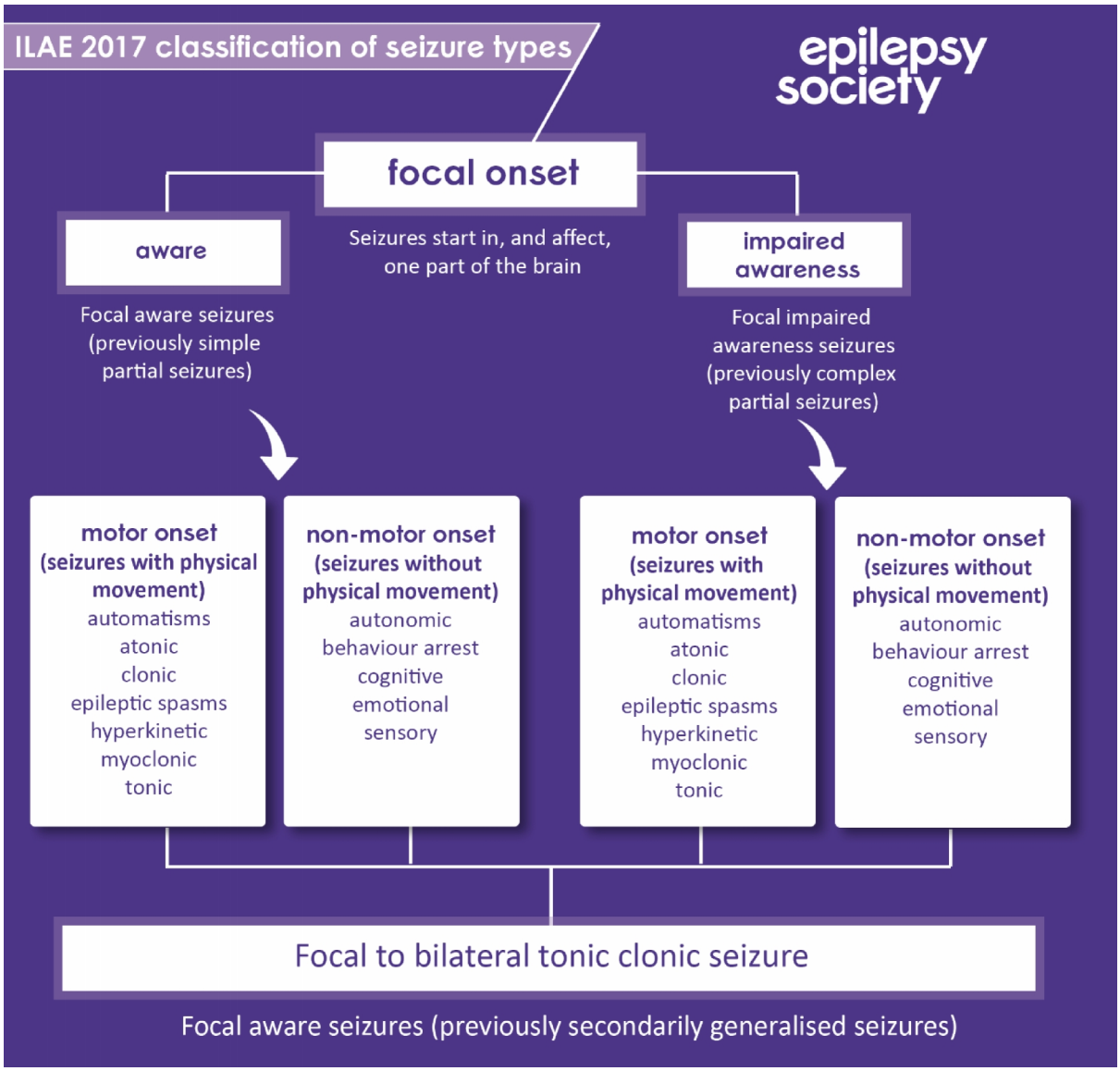
- Non-motor

    i- Behaviour arrest

- UNCLASSIFIED

This new classification is designed to be flexible and allows the use of other descriptive terms. Even though most seizures can be classified on the basis of signs and symptoms during the episode, other information in the form of EEG, MRI or other imaging, videos, blood tests, genetic tests are also helpful.

FOCAL SEIZURES



They arise from a discrete neuronal network[11] or a broader one which is still localized to one side of the brain. The older subcategories of simple and complex have been eliminated and current terminologies are based on the presence of awareness as described above. The routine EEG obtained in between seizures is often a normal variant or might show a discharge pattern called epileptiform spikes or sharp waves. Occasionally it can be even non-localising if it originates from the inferior frontal lobe or medial temporal lobe, which are far from the scalp electrodes.

Further subcategories depend on the motor or non-motor behaviour of the seizure. Motor seizure arises from primary motor cortex and affects that body part depending on the location in the homunculus, for example, near the area controlling hand movement in right side causes left-hand involuntary movements characterized by typical clonic movements or repetitive flexion and extension movements with a frequency of ~ 2-3 Hz. This cortical region controlling hand movements are adjacent to that of facial expression which explains the synchronous face movements. The focal nature can be visualized using an ictal EEG which will demonstrate the restriction of seizure activity to a limited area if it is involving the cerebral convex.

Some eponymous features limited to focal motor seizures are “Jacksonian march” and Todd’s paralysis. John Hughlings Jackson, the father of modern epileptology described the phenomenon of motor movements beginning in a restricted area like fingers and gradually progressing to a larger area of the extremity due to the spread of seizure activity. The latter term characterizes the

localized paresis experienced by the patients involving the affected region lasting from minutes to hours. *Epilepsia partialis continua,* when the seizure may continue for hours or days, is often refractory to medications.

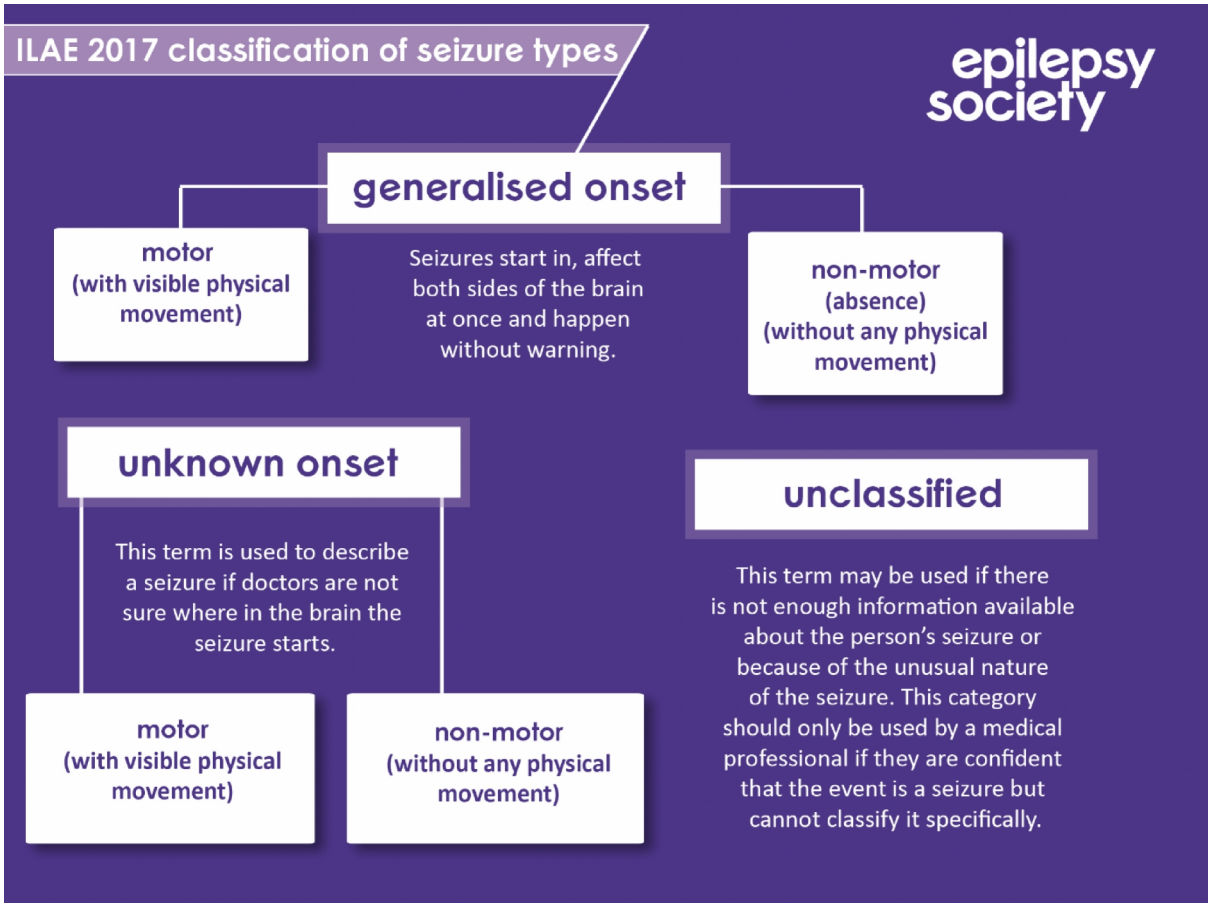
Non- motor seizures may manifest as changes in sensation, equilibrium, vision or autonomic function. Depending on the cortical area involved there will be alterations in the sensations or even higher functions resulting in fear, detachment, depersonalization, déjà vu, macropsia which are referred to as auras. But this term has been eliminated from current classification.

Automatisms refers to automatic involuntary movements ranging from simple chewing or lip-smacking to elaborate behaviours like running.

FOCAL TO BILATERAL TONIC-CLONIC

The evolution of a focal seizure to involve both lobes is most frequently seen when the foci are in the frontal lobe, though it can be seen elsewhere also. This is a difficult diagnosis to differentiate from primary GTCS as the initial subtle symptoms may have escaped the notice of the bystanders. Nonetheless, this distinction is sine qua non as the further management is entirely different.

GENERALIZED SEIZURES



They are thought to arise at some point but rapidly spread to involve both sides of brains. It includes many variants like absence, GTCS, atonic and myoclonic seizures.

Typical absence seizures have characteristic sudden onset lapse of consciousness while retaining posture, lasting for a few seconds, without any postictal confusion. Subtle motor signs usually accompany this, for example, chewing, rapid eye blinking or low amplitude hand movements. Typical absence seizures usually have a genetic predisposition and are common in the first decade of life. The EEG hallmark is a symmetric, generalized 3-Hz spike and wave discharge superimposed on a normal background.

Atypical absence seizures are both clinically and electro physiologically different from a typical one and the EEG shows a generalized slower wave pattern of < 2.5 Hz. They result from multifocal structural anomalies in the brain parenchyma causing other signs like mental retardation and are thus less responsive to conventional treatment.

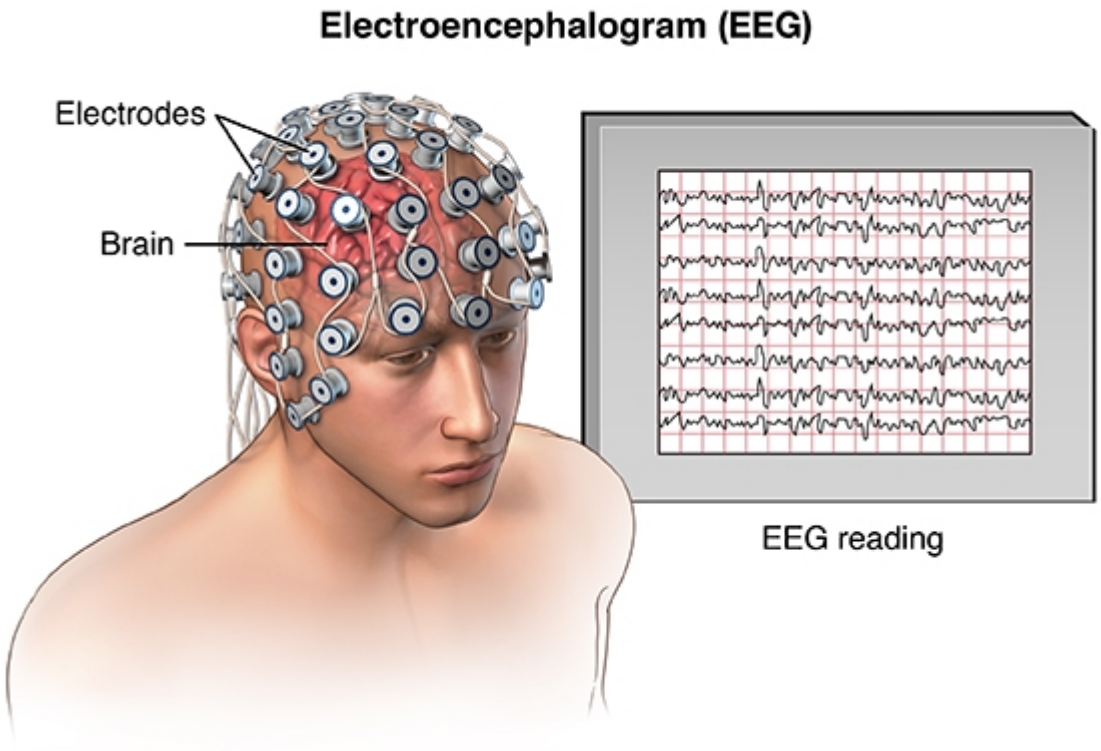
Generalized tonic-clonic seizures account for ~ 10% of all cases of epilepsy. It is the most common variant resulting from metabolic insults. Other than being generalized, they also differ from focal variant by abrupt onset without any warning signs or characteristic aura most of the time. Initial tonic contraction of all muscles involving respiratory muscles results in an 'ictal cry' and tongue biting. This is followed by a clonic phase of initial mild generalized tremors rapidly giving way to violent flexor spasms. This phase also characterized by prominent autonomic signs like tachycardia, hypertension, dilated pupils etc. The culmination of the episode takes the patient into a deep coma. The patient recovers after a few minutes confused with no recollection of the episode. The EEG typically shows repetitive spike-wave discharges followed by 10s periods of 10 Hz spikes[12]. Spikes then begin to mix with slow waves giving the EEG a polyspike and wave pattern during the clonic phase.

Myoclonic seizures are typical brief muscular contractions that might involve only one muscle or a part of it but can occasionally be large to cause limb displacements or trunk muscles. They are caused by cortical dysfunction which marks the EEG with synchronous spike and wave discharges with the myoclonus.

Atonic seizures present with abrupt loss of postural muscle tone resulting in a fall, with brief impairment of consciousness but no postictal confusion. This puts the patient at risk of other injuries like head injury, burns etc. The EEG shows brief discharges immediately followed by slow waves characteristic of loss of muscle tone.

ELECTROPHYSIOLOGY OF SEIZURES

The cerebral cortex generates spontaneous electrical activity due to currents flowing in the extracellular spaces arising from the synaptic potentials of neurons, which is recorded in an EEG. This cortical activity is influenced by subcortical structures especially thalamus and reticular formation. These brain rhythms are amplified in an EEG as a waveform with a frequency range of 0.5 to 30 Hz.



An established focal seizure is characterized by an EEG spike due to near simultaneous firing of a large number of neurons across a large surface of the cortical region. This starts at a small discrete region slowly spreading across a larger area. The influx of extracellular calcium results in a long-lasting neurilemmal depolarisation, leading to voltage-gated sodium channel opening which generates repetitive action potentials. Hyperpolarization after potential follows this, mediated by either GABA receptors or potassium channels. The seizure wave front thus created is slowed and stopped by intact hyperpolarisation of the inhibitory neurons by feed forward inhibition. But, when a vast number of adjacent neurons are recruited with sufficient activation, via synaptic and non-synaptic mechanisms, excitatory currents propagate into contiguous areas and even distant areas via commissural pathways. Abnormalities in the oscillatory rhythm between thalamus and cortex, normally produced in sleep account for absence seizures. But the limited knowledge of the brain connectivity at system levels keep the mechanisms for most of the generalized seizures still in the dark. Structural changes in the neuronal network transform it into a chronically excitable one, referred to as epileptogenesis.

AETIOLOGY OVERVIEW

Aetiology of seizures in developing countries varies from that of developed countries. It varies vastly depending on the age group, geography, socio-economic status, medical setting etc. Genetic causes are common in the first decade whereas perinatal insults and metabolic causes contribute towards seizures in infancy. Similarly, trauma, inherited predisposition and drug abuse are common in young

adults in contrast to malignancy, degenerative disorders and stroke in elderly. In India, neurocysticercosis is very common which can be used as a 'biological marker' for the socio-economic status of the region[6]. 40% of focal seizures are caused by them with higher prevalence in Punjab, Haryana, U.P., and Delhi. Malignancy is common after 30 years of age and causes epilepsy in 10%. Non-epileptogenic foci like brainstem and cerebellum are the most common sites of malignancy in children reflecting the lower incidence in children. Metabolic abnormalities including electrolyte disturbances and drug abuse are another important cause.

AETIOLOGY ACCORDING TO AGE

Neonates

- Perinatal hypoxia and ischemia
- Acute CNS infection
- Intracranial hemorrhage and trauma
- Metabolic disturbances
- Developmental disorders
- Drug withdrawal
- Genetic disorders

Infants and children (<12 years)

- Febrile seizures
- CNS infection
- Genetic disorders

- Trauma
- Developmental disorders
- Idiopathic

Adolescents (12-18 years)

- Genetic disorders
- Trauma
- Illicit drug use
- Infection
- Idiopathic
- Brain tumour

Young adults (18-35 years)

- Alcohol withdrawal
- Trauma
- Brain tumour
- Infection
- Illicit drug use
- Idiopathic

Older adults (>35 years)

- Brain tumor
- Alcohol withdrawal
- Cerebrovascular accidents
- Infection

- Degenerative disorders like Alzheimer's disease
- Metabolic disorder
- Idiopathic

The acute symptomatic seizure has an underlying medical condition or neurological illness but should be termed so only if there is a close temporal association with the insult, be it systemic, metabolic or toxic.

Metabolic derangements

- Hyponatremia
- Hypernatremia
- Hypoglycemia
- Hyperglycemia
- Hyperosmolarity
- Hypocalcemia
- Respiratory alkalosis

Drug-induced seizures

- Isoniazid, penicillins
- Theophylline, aminophylline
- Meperidine
- Lidocaine
- Amitriptyline, imipramine, fluoxetine, doxepin, maprotiline
- Ephedrine, terbutaline, phenylpropanolamine
- Ketamine, halothane, enflurane, methohexital

- Haloperidol, trifluoperazine, chlorpromazine
- Cyclosporine
- Methotrexate, asparaginase
- Cocaine, phencyclidine, amphetamines
- Alcohol withdrawal

Illnesses

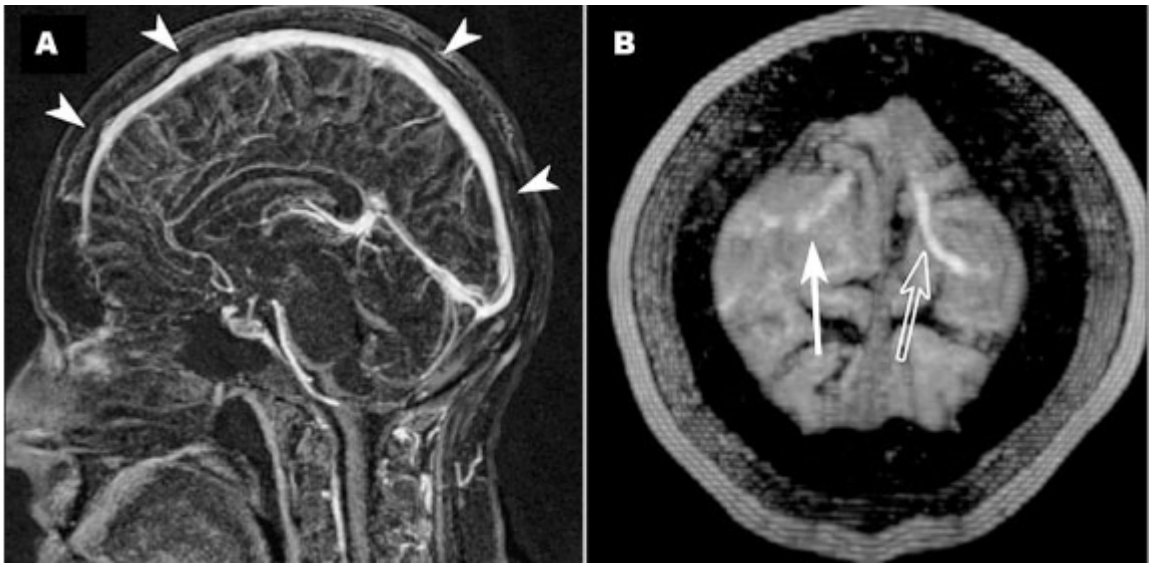
- Hypertensive encephalopathy
- Polyarteritis nodosa
- Eclampsia
- Liver failure
- Syphilis
- Porphyria
- Whipple's disease
- Sickle cell disease
- Systemic lupus erythematosus
- Renal failure
- Thrombotic thrombocytopenic purpura

Neurologic conditions

- Encephalitis
- Stroke
- Brain tumor
- Meningitis

- Head trauma
- Brain abscess

Seizures are common in stroke especially in the early phase with a frequency up to 2.5- 5.7% within 2 weeks[13]. Studies have shown that the type of stroke doesn't affect the risk of early seizures. Acute seizures in stroke are uncommon in young but incidence increases after the age of 45 years. Haemorrhagic stroke in the cortex has increased susceptibility for development of epilepsy[14]. Similarly, a micro vascular disease involving the CNS also cause epilepsy. Cortical sino-venous thrombosis is also associated with early seizures at a higher frequency.

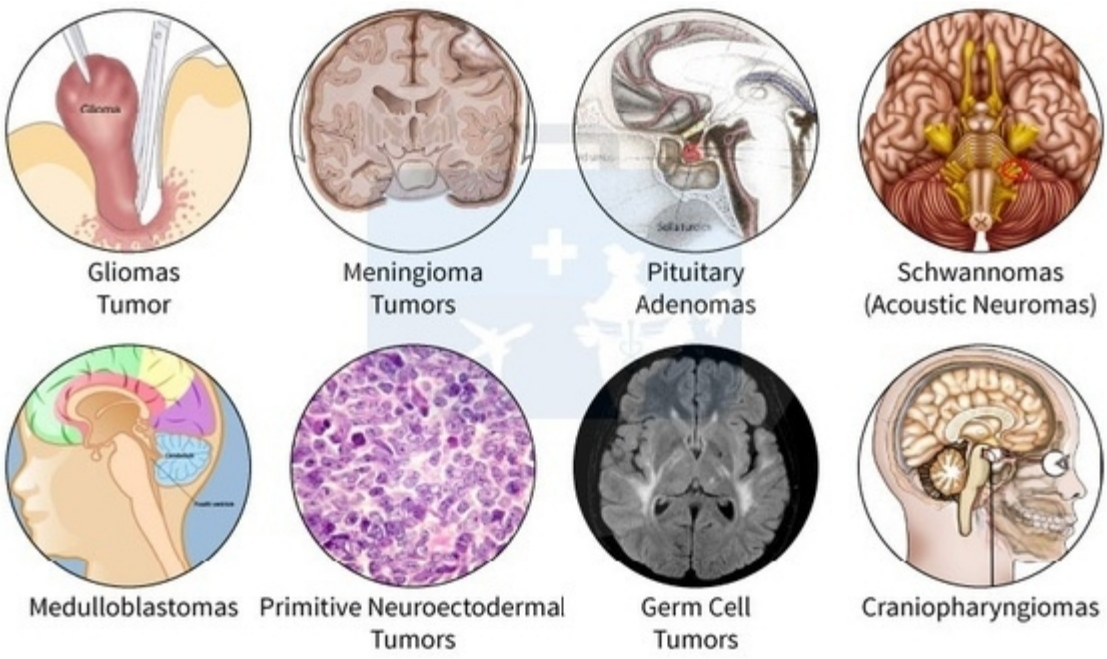


MR venography: sagittal and axial views showing cerebral venous thrombosis

The incidence and prevalence of epilepsy and seizures increase with advancing age, it forms the second peak of the bimodal age distribution, with an annual incidence of 85 per 100000 in the age group of 65-69 years and 159 per 100000 among those over 80 years[15]. There are a variety of causes for seizures in elderly

with the most reported cause being cerebrovascular accidents. They account for 30-50% of all aetiologies in elderly. Strokes lead to seizures from areas that are partly destroyed rather than complete infarction[14].

Primary neurodegenerative disorders account for 10-20 % of cases with the most common being Alzheimer's disease. The type of neurodegenerative disorder, its severity, dementia, race and epileptiform discharges all contribute to seizures and epilepsy. Trauma, yet another important cause in the elderly, is quite common due to frailty and likeliness to fall. Seizures are the presenting complaint in 20-40% patients with brain tumors, which is the second most common cause of epilepsy in the elderly. Common tumors associated with this scenario are primary CNS lymphoma, meningioma, anaplastic ependymoma, and astrocytoma.



Types of brain tumour

Metabolic abnormalities cause 9% of acute onset seizures[6]. These include uremia, hypoglycemia, hyperglycemia, and withdrawal from sedative-hypnotic agents. Seizures may be the only manifestation of electrolyte disturbances like sodium level alterations, hypocalcemia (<5.0 mg/dL), hypomagnesemia (<0.8 mg/dL)[14]. Magnesium influences neuronal excitability by stabilizing membranes via altering the calcium mobilization. In a study from south India, Joseph et al reported a fall in bicarbonate levels, hypochloremia, and hyponatremia as the most common blood findings in epileptic patients[16].

Drug and toxin-induced seizures[17], need aggressive management to have a significant impact on the outcome. It is estimated to account for 6.1% of new onset seizures[18]. Exposure as well as withdrawal from either result in seizures. Seizures due to intoxication with organophosphorous compounds and camphor are prevalent in children than in adults[19],[20]. The majority are harmless and self-limited unless untreated prolonged seizing results in irreversible brain injury. Standard anticonvulsant drugs are the mainstay of management.

Acute symptomatic seizures are provoked seizures which result from any insult to the brain. They have a lower risk for subsequent unprovoked seizures with recurrence rates of 10-20 %[5]. The common causes include severe head injury, acute stroke, subarachnoid hemorrhage, brain surgery and CNS infections.

Alcohol overuse, as well as withdrawal, causes seizures. The most commonly used questionnaire is CAGE[21], to assess the level of alcohol consumption and dependence. The other alternatives include the Alcohol Use Disorders Identification Test (AUDIT), Brief Michigan Alcoholism Screening Test (Brief

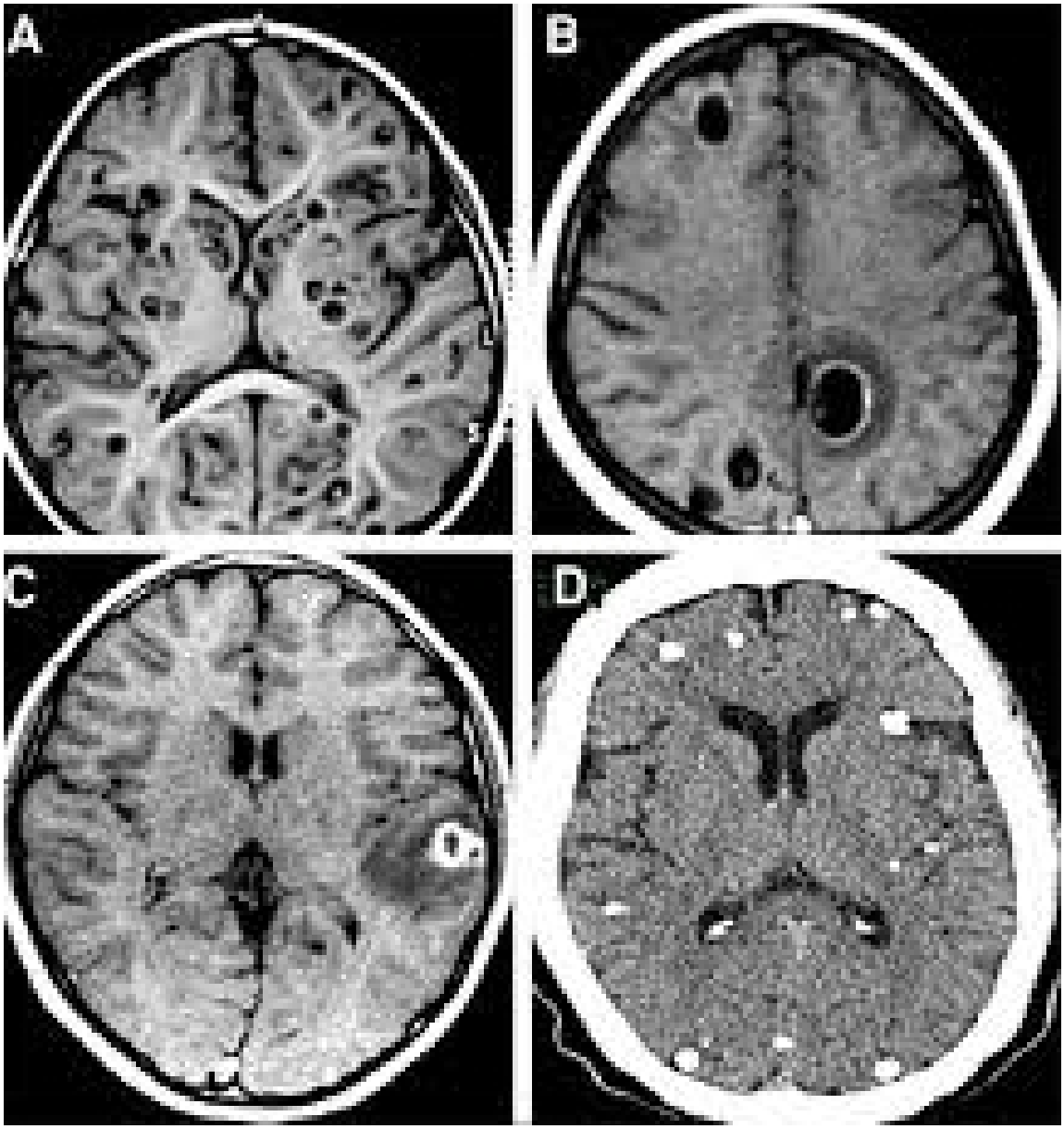
MAST), Munich Alcoholism Test (MALT). Alcohol overuse seizure has a high incidence of a structural intracranial lesion as seen in CT whereas EEG abnormalities are lower in such patients.

Eclampsia, with an incidence of 1 in 2000-3000 pregnancies in the west[22], remains an unpredictable cause of life-threatening complications during pregnancy. It is defined as the occurrence of unexplained seizure in women with preeclampsia (hypertension with proteinuria) in pregnancy. Although the mechanism is currently unknown, most accepted hypotheses are regarding the 'over autoregulation' of cerebral perfusion resulting in ischemia and that of a hypertensive encephalopathy resulting in vasogenic oedema. Cerebral venous sinus thrombosis accounting for 1% of ischemic cerebrovascular diseases[23], present commonly with seizures in up to 33% of cases. Focal neurologic deficits and thrombosis of superior sagittal sinus were found to be independent risk factors for seizures.

Anti-epileptic drugs in the first trimester of pregnancy increase the risk of major congenital malformations, the most noted one being spina bifida with valproic acid and carbamazepine[24].

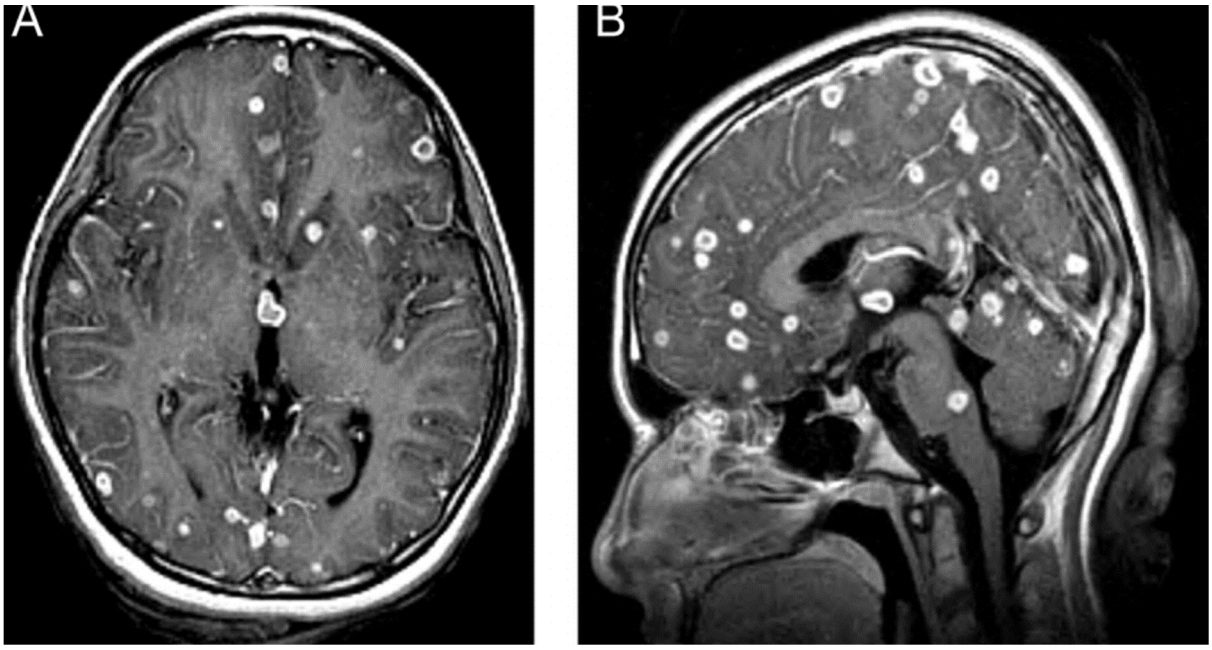
Due to the poor socio-economic status, infections are a leading cause of seizures in developing countries. Single, small enhancing CT lesions (SSECTL) is defined as a disc or ring measuring <20 mm and enhances on contrast administration[24]. It usually represents dying cysticerci. Neurocysticercosis, common in *Taenia solium* endemic regions, most commonly presents with seizures[25]. Imaging is instrumental in clinching a diagnosis. Actively degenerating

parenchymal cysticerci in the brain (single or multiple), calcified lesions consistent with inactive cysticerci, or both the above combined is highly suggestive of the diagnosis. But to establish it as the cause of seizure anatomical location of the lesion and electroclinical localization of the seizure onset are important. Due to the poor sensitivity of serology, especially in single lesions, it is not considered mandatory. But strong seropositivity is associated with a higher risk of seizures[26] The common locations in frontal and parietal lobes account for the focal seizures with aphasia seen exclusively in neurocysticercosis in some studies[25]. And theoretically, the diffuse location of multiple lesions can result in multiple varieties of seizures. It is conjectured that the phasic release of antigens results in cerebral inflammatory response resulting in seizures which are clustered over a small period of time. These cluster seizures though not specific to NCC, has been found to be significantly more common in this scenario.



CT Brain in a case of neurocysticercosis

Hematogenous spread of tubercle bacilli result in parenchymal tuberculoma, most common in the posterior fossa[27] and often lack a history of infection. An avascular mass lesion of low density with exaggerated local oedema is suggestive of tuberculoma in CT. They become encapsulated with ring enhancement in later stages.

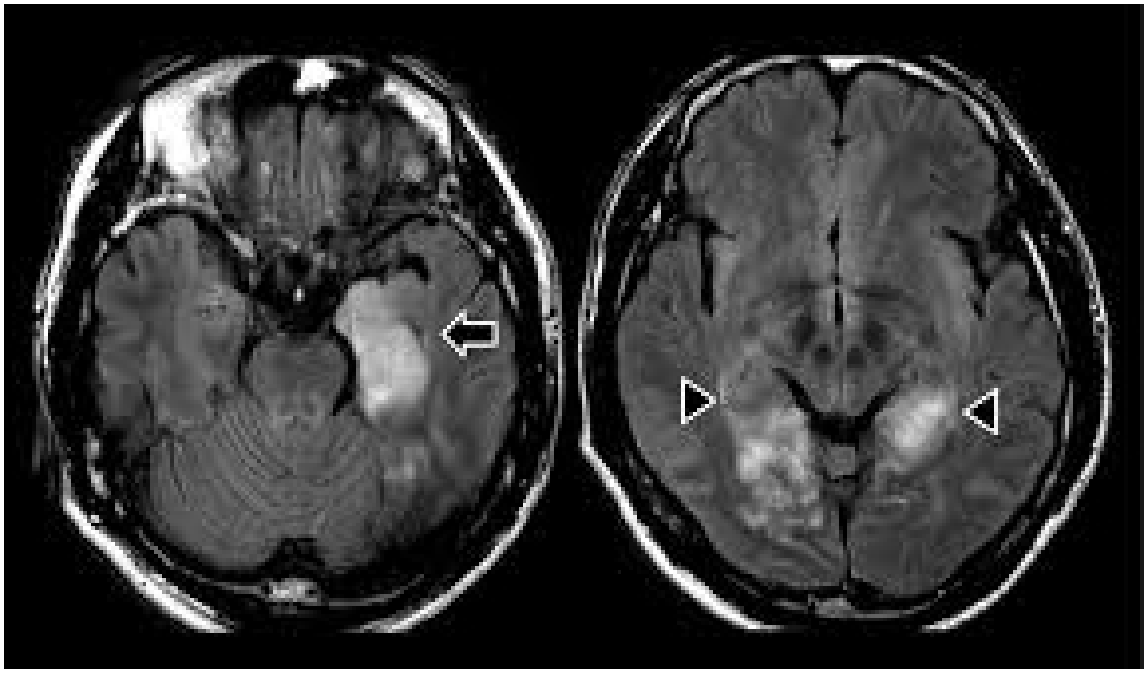


MRI Brain in a case of tuberculoma

The incidence of T.B is more in HIV patients due to immunosuppression and those with low CD_4 counts experience a paradoxical worsening of tuberculosis with the initiation of ART due to the recovery of the immune system, which is termed Immune Reconstitution Inflammatory Syndrome[28]. Tuberculoma causes mass effect or forms an abscess, resulting in seizures. T.B meningitis has been reported to have a varied incidence ranging from 17-93% with the multi-factorial cause of seizure induction like cerebral edema, increased ICP, hydrocephalus, meningeal irritation, tuberculomas and cerebral infarction[29].

Seizures in meningitis are often associated with poor outcome and usually presents within 24 hours[30]. Focal seizures are due to edema or ischemia whereas generalized seizures and even status epilepticus result from the associated fever, metabolic derangement, spread from a focal onset and antimicrobial toxicity. In a study about viral encephalitis in North India[31], it was reported that Herpes Simples

Encephalitis has more severe and frequent seizures in up to 75%, followed by Japanese Encephalitis (54%) and dengue (23%).



MRI Brain in herpes simplex encephalitis

CNS involvement is noted in 90% of AIDS patients at autopsy and new onset seizures up to 20% have been documented in some studies[24]. Cryptococcal meningitis, toxoplasma encephalitis, CNS tuberculosis, AIDS dementia complex and progressive multifocal leukoencephalopathy are a few conditions found in such patients. In addition, neurotropic nature of the HIV itself is harmful to CNS, resulting in seizures.

Dengue virus is a global threat with increased incidence in tropical countries like India. Dengue virus causes a spectrum of neurological manifestations ranging from meningitis, encephalopathy to GBS. The virological characteristics of dengue have been changing in recent years resulting in increased

neurological complications[32]. Dengue encephalitis should be suspected in any case of seizures with altered sensorium with a history of febrile illness and thrombocytopenia.

Idiopathic generalized epilepsy, which constitutes upto one third of all epilepsises in some studies, have a genetic predisposition. It is characterised by an early onset seizure with a normal EEG background associated with paroxysms of generalised EEG discharges exacerbated by photosensitivity and hyperventilation.

Post-traumatic seizures have an incidence of 4-53%. In a study by Thapa et al[33], several risk factors were analysed which included age <10 years, female sex, amnesia>30 min, fall from a height, GCS at presentation, brain oedema etc. They are classified into early and late onset, based on the time of occurrence before or after 1 week[34]. It portends a worse functional outcome after the brain injury. Glycerol, a marker for cell membrane breakdown[33] is elevated suggestive of additional membrane injury following seizures.

RARE ETIOLOGY

Reflex epilepsies are in response to a particular sensory stimulus. A classical example is Hot Water Epilepsy which occurs after pouring hot water overhead, particularly seen in south India during the winter season[24]. Several factors like genetic, environmental and patient habits have been associated with it, despite no clear knowledge of the mechanism. It has been proposed that an aberrant thermoregulatory system resulting in sympathovagal imbalance might be the cause.

An autosomal dominant seizure syndrome characterized by a triad of cortical tremor, GTCS and multifocal myoclonus, known by the acronym FAME, Familial Adult Myoclonic Epilepsy, has recently caught attention. It is a nondisabling, slowly progressive condition linked to *FAME I* locus in chromosome $8q^{35}$ seen in age group 12-50 years. Most studies have shown a generalized polyspike wave with a photo myoclonic response. Here, seizures are commonly seen in 1-2 hours of sleep, mostly GTCS.

Multiple cases of Eating epilepsy have been reported from south India and Sri Lanka. Imaging studies have shown perisylvian lesions which might be affecting complex neuronal circuits around it.

Lafora body disease due to mutation of the EPM2A gene on chromosome 6q and EPM2B gene. Epilepsy Progressive Myoclonus gene Type 2A codes for the laforin protein, which is involved in neuronal migration during early brain development.

Progressive myoclonal epilepsy, a disease complex of progressive myoclonus, ataxia, cognitive impairment and other neurological impairments. It is caused by multiple conditions namely, Lafora body disease, myoclonic epilepsy with ragged red fiber syndrome (MERRF), dento-rubro-pallidal atrophy, Unverricht-Lundborg disease, sialidoses, neuronal ceroid lipofuscinosis etc.

INITIAL EVALUATION

Evaluation of a patient with new-onset seizures should take place in an orderly manner. Distinguishing the current event from other paroxysmal

conditions mimicking seizures is the initial step of management. These include transient ischemic attack, migraine, syncope or psychogenic non-epileptic seizures[5]. Clinical evaluation with a focus on history, patient's experience, awareness, and recollection is instrumental in clinching the diagnosis of a seizure.

Subjective information on prodromal symptoms like aura is important in localizing. Further assessment of the previous history of subtle symptoms like myoclonic jerks, staring spells etc. might point towards the diagnosis of specific epilepsy syndromes. The patient's and witness' accounts recorded separately increases the accuracy of the report.

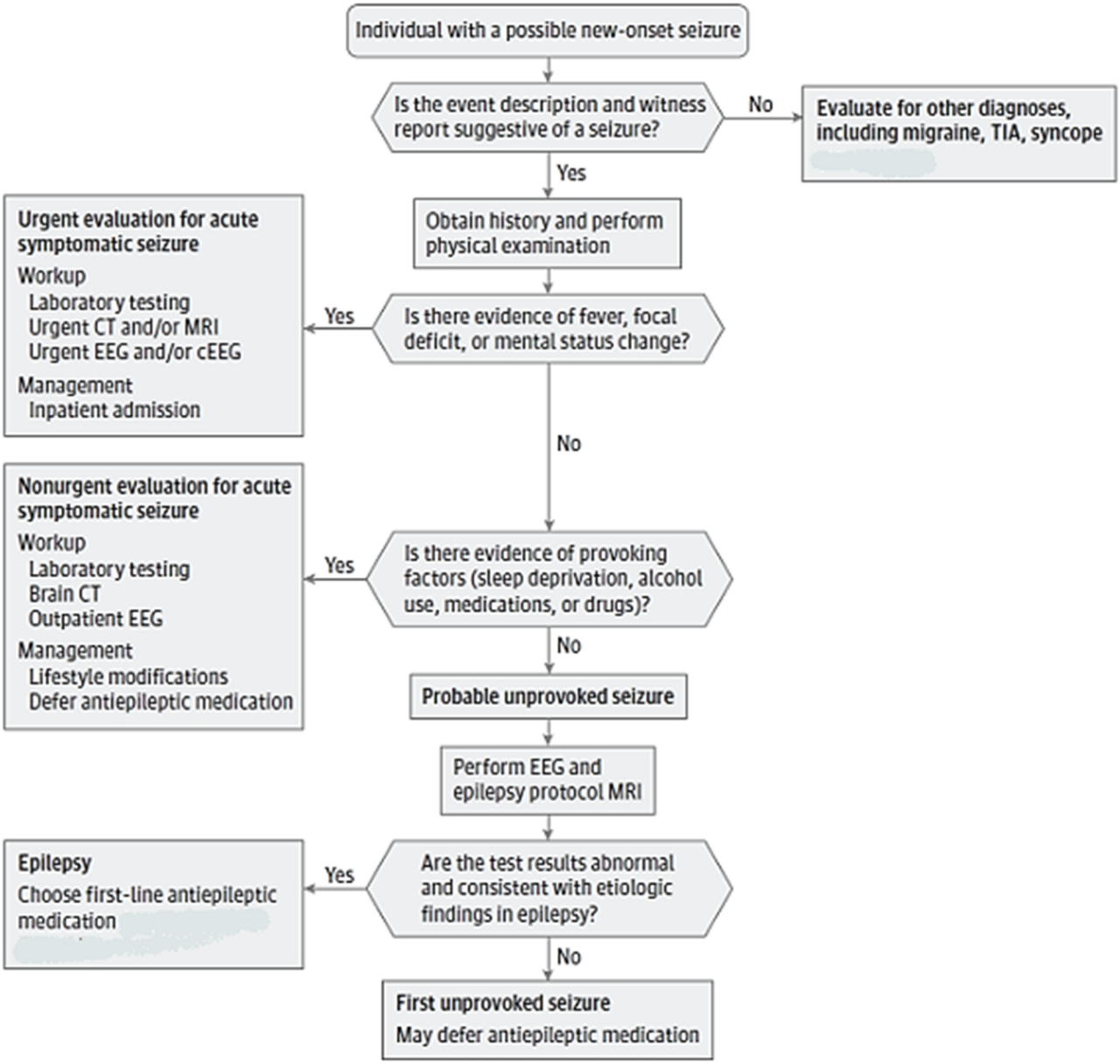
Physical examinations like tongue bites can distinguish epileptic seizures from psychogenic non-epileptic seizures. The resultant fall might result in bruises and scrapes. The finding of neck rigidity or asterixis suggests an underlying systemic disorder. Next concern is, assessing for the presence of provocative factors, the absence of which calls for an exquisite workup to check for risk of recurrence.

This workup includes both structural and functional imaging. Brain imaging revealed parenchymal anomalies in 10% of cases using CT which improved up to 30% with MRI[5]. However, due to ease of access, CT is usually the first imaging done. An epilepsy protocol-specific brain MRI uses thin 1-3 mm slices with coronal FLAIR sequence which confers additional sensitivity over normal MRI. These are useful in identifying lesions usually missed in CT like low-grade glioma, hippocampal sclerosis, cavernous and cortical malformations.

Standard EEG monitoring is done for 30 minutes after a new onset seizure, to evaluate its type. Its yield increases when done within 24-48 hours after the onset. It is indicated as emergent investigation if a patient doesn't come back to baseline neurological function within 30-60 minutes of the end of seizure or has altered consciousness or neurological dysfunction.

Metabolic causes like uremia, hyponatremia, hypoglycemia, drug toxicity should be checked in blood, especially in acute symptomatic seizures besides serum prolactin (which differentiates from a psychogenic seizure)[36]. Lumbar puncture is an invaluable component in the workup of suspected infectious causes with altered sensorium and leucocytosis. Blood culture, CSF culture, and CSF for viral PCR in suspected HSV encephalitis also needs to be done. The essential diagnostic procedures can be summarised as[37]

- Clinical examination
- Assessment of seizure semiology
- Lab tests- routine, CSF and Drug screening
- Early standard EEG
- Sleep deprived EEG in 1 week
- High-resolution MRI.



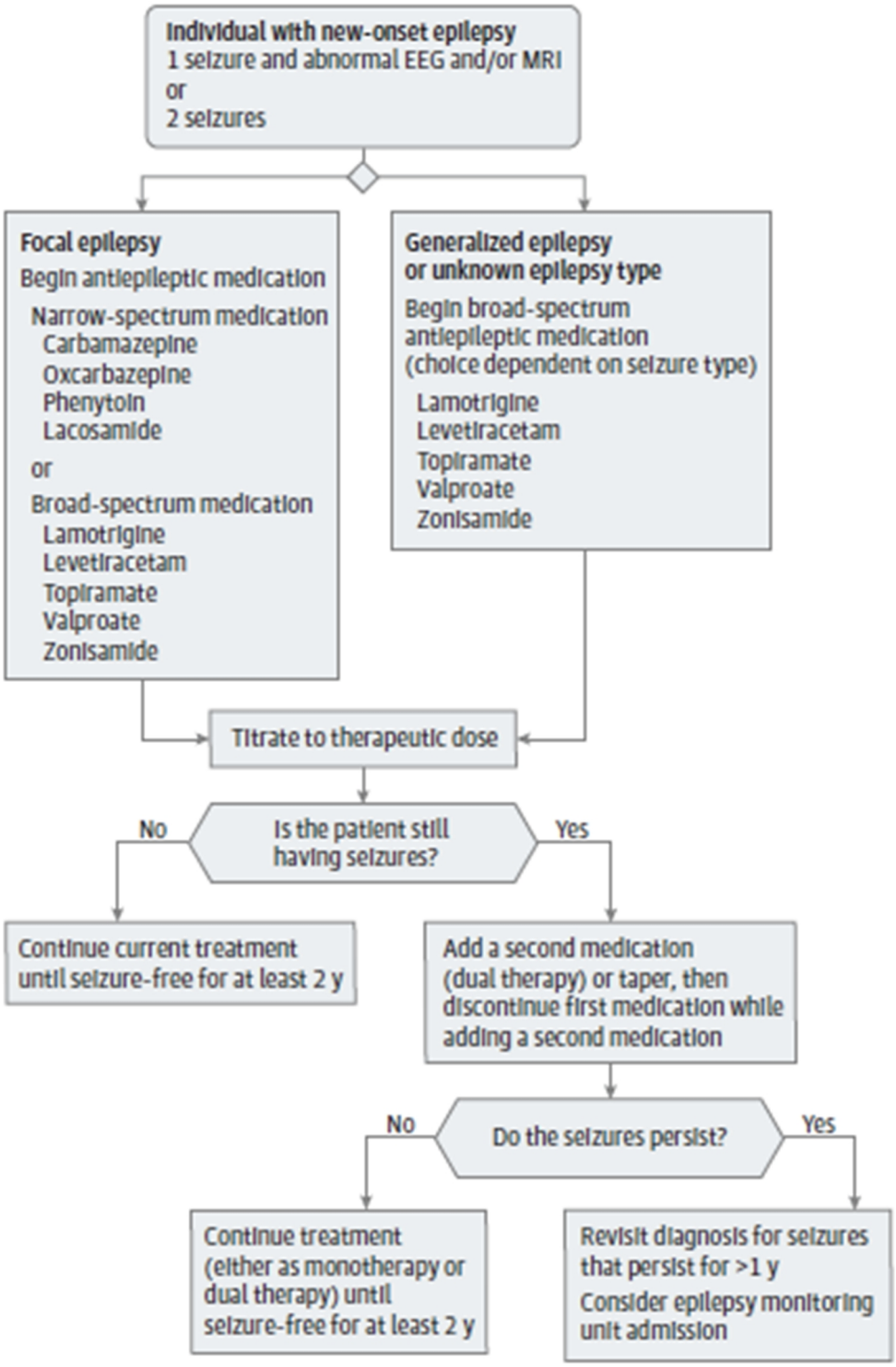
Another important consideration is to distinguish an unprovoked seizure from an epilepsy syndrome, especially while considering drug treatment. Though the diagnosis of epilepsy can't be made after a single seizure, an epileptic syndrome can be diagnosed after one with the help of clinical details, family history, EEG, and MRI.

TREATMENT

Suspected new-onset seizures in adults after the first episode can be safely managed as an outpatient[38]. Admission is reserved for those with persistent seizures or other signs, abnormal investigations etc. Further evaluation with the specific investigation is done in first seizure clinic. The risk of recurrence after a first unprovoked seizure was found to be 42% over the next two years, in a meta-analysis[37]. More than half of these occur within six months and falls exponentially thereafter. Studies have found that restriction of recreational activity for two to three months is adequate whereas driving is not permitted for either one or ten years depending on whether it is commercial or not.

Anti-epileptic drug treatment is not indicated in all cases. Nonetheless, it reduces the risk of seizure recurrence, rest alone eliminate it. Hence the initiation of the same must be thoroughly weighed against the risk of adverse reactions from chronic drug use. Drug choice should be individualized considering drug reactions, teratogenicity, cognitive abilities, and cost. There are no published data regarding the duration of treatment in adults[37], hence decisions have to be individualized

Among the three stages of prevention, most of the treatment involves tertiary prevention. In a recent critical review of the prevention task force of ILAE[39] regarding primary prevention, it was found that effective intervention by public departments regarding maternal and child health care, brain injury prevention, strike prevention could reduce the burden of epilepsy as these are the most preventable causes of new-onset seizures.


Individual with new-onset epilepsy
1 seizure and abnormal EEG and/or MRI
or
2 seizures
Focal epilepsy
Begin antiepileptic medication
Narrow-spectrum medication
Carbamazepine
Oxcarbazepine
Phenytoin
Lacosamide
or
Broad-spectrum medication
Lamotrigine
Levetiracetam
Topiramate
Valproate
Zonisamide
Generalized epilepsy
or unknown epilepsy type
Begin broad-spectrum
antiepileptic medication
(choice dependent on seizure type)
Lamotrigine
Levetiracetam
Topiramate
Valproate
Zonisamide
Titrate to therapeutic dose
Is the patient still
having seizures?
No
Yes
Continue current treatment
until seizure-free for at least 2 y
Add a second medication
(dual therapy) or taper, then
discontinue first medication while
adding a second medication
Do the seizures persist?
No
Yes
Continue treatment
(either as monotherapy or
dual therapy) until
seizure-free for at least 2 y
Revisit diagnosis for seizures
that persist for >1 y
Consider epilepsy monitoring
unit admission

MATERIALS AND METHODS

Source of data- Patients admitted to Tirunelveli Medical College during the period of study were taken for the study considering the inclusion and exclusion criteria.

Method of collection of data – Informations were collected through prepared proforma.

Duration of study – March 2017 to March 2018

Type of study – Prospective observational study

Sample size – Adult patients admitted with new-onset seizures in the hospital.

Inclusion criteria

- New onset seizures
- Age > 13 years

Exclusion criteria

- Known case of seizure disorder
- Movement disorders
- Transient ischemic attacks
- Psychogenic seizures
- Road traffic accidents
- Mental retardation
- Age <13 years
- Narcolepsy

- Insufficient clinical data for diagnosis

Methodology

The study was conducted in the department of general medicine, Tirunelveli Medical College from March 2017 to March 2018. Institutional Ethical Committee approval was obtained for the research proposal. Nature, methodology, and risks involved in the study were explained to the patients and informed consent was obtained. All the information collected was kept confidential and the patient was given full freedom to withdraw at any point during the study. All provisions of the Declaration of Helsinki were followed in this study.

All patients who qualified the inclusion criteria were evaluated. They were interviewed as per the proforma and a complete clinical examination was done. Cases of new-onset seizures were diagnosed with clinical history, examination, laboratory and radiological studies.

Parameters studied

- Haemoglobin, total count, differential count, platelet count.
- Random blood sugar
- Renal function test
- Liver function test
- Serum electrolytes (Na+, K+, Ca2+)
- Dengue serology (if needed)
- Electroencephalogram

- CT brain and MRI (if needed)
- ECG

Statistical analysis

The analysis was done mainly using simple percentage analysis. Descriptive analyses were reported as mean and standard deviation of continuous variables. SPSS version 21.0 was used.

OBSERVATION & RESULTS

A total of 206 patients with new onset seizures were enrolled for the study. Their age ranged from 13 years to 85 years.

TABLE 1: AGE DISTRIBUTION OF PATIENTS WITH NEW ONSET SEIZURES

S.NO	AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
1	13-20	35	17%
2	21-30	49	24%
3	31-40	37	18%
4	41-50	29	14%
5	51-60	23	11%
6	61-70	21	10%
7	71-80	10	5%
8	>80	2	1%

Highest incidence was in the age group of 21 to 30 years followed by 31 to 40 years. Age above 60 years accounted for only 16% of the cases.

FIGURE 1: AGE DISTRIBUTION OF PATIENTS WITH NEW ONSET SEIZURES

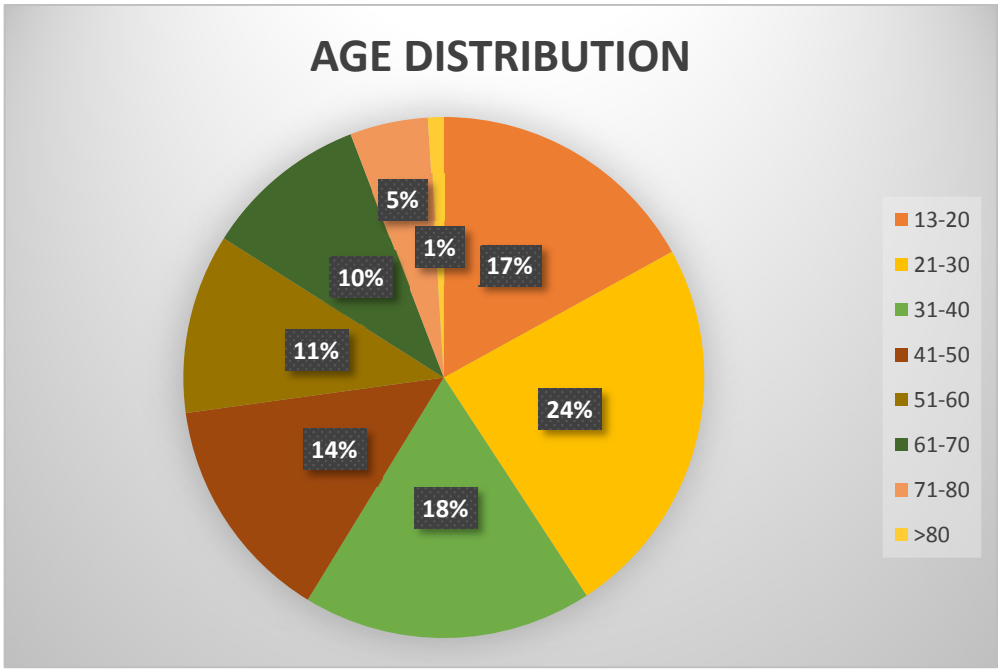


TABLE 2: SEX DISTRIBUTION OF PATIENTS WITH NEW ONSET SEIZURES

S. NO	SEX	NO OF PATIENTS	PERCENTAGE
1	MALE	113	55%
2	FEMALE	93	45%

The study group contained more males than females in the ratio of 1.2:1.

TABLE 3: AGE VS SEX DISTRIBUTION

S.NO	AGE IN YEARS	MALE	FEMALE
1	13-20	16	19
2	21-30	23	26
3	31-40	20	17
4	41-50	18	11
5	51-60	15	8
6	61-70	10	11
7	71-80	9	1
8	>80	2	0
	TOTAL	113	93

FIGURE 2: AGE VS SEX DISTRIBUTION

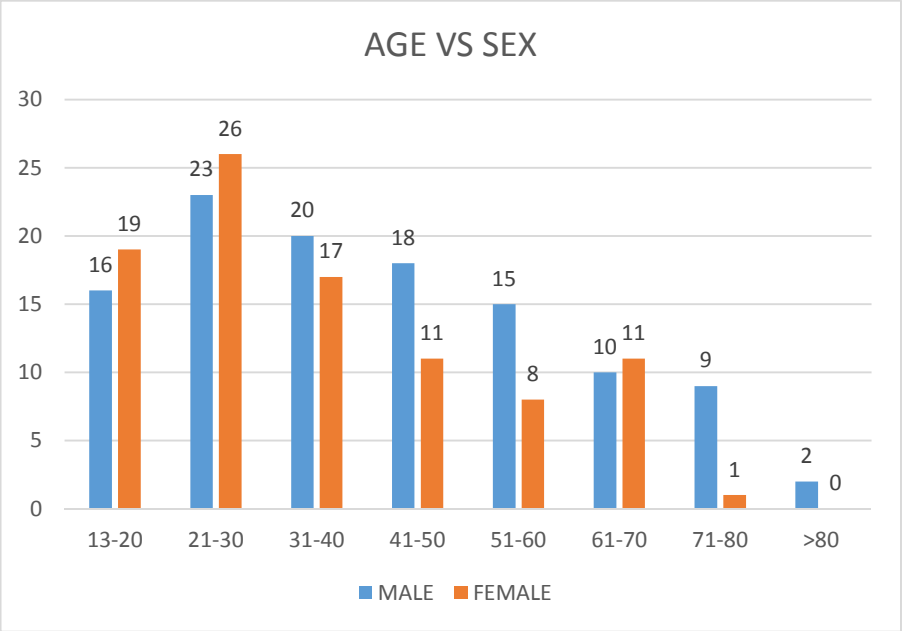
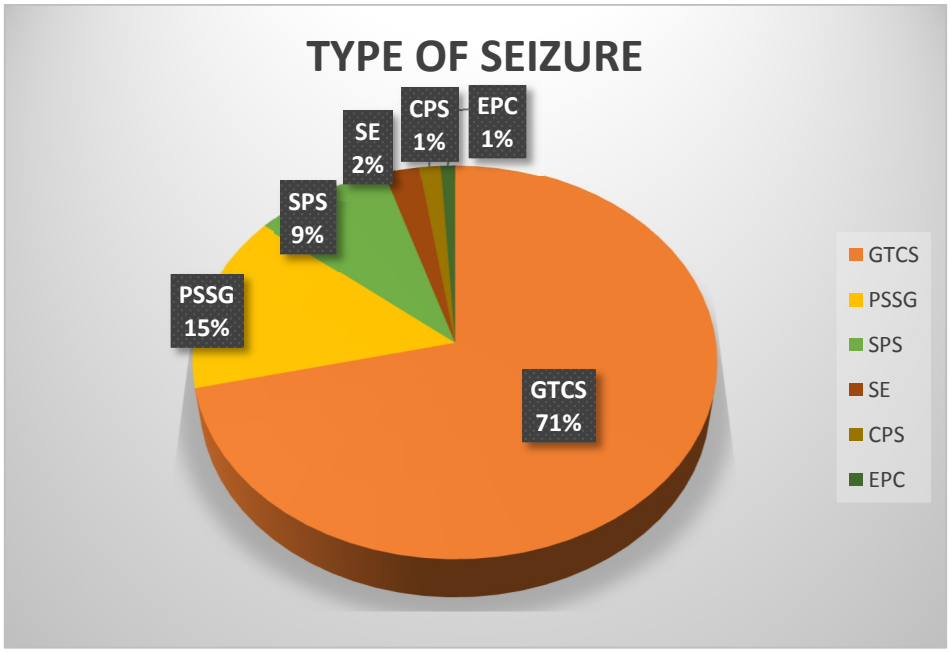


TABLE 4: DISTRIBUTION OF TYPE OF SEIZURES

S. NO	TYPE OF SEIZURE	NO OF PATIENTS	PERCENTAGE
1	GENERALISED TONIC CLONIC SEIZURES	147	71%
2	PARTIAL SEIZURES WITH SECONDARY GENERALISATION	30	15%
3	SIMPLE PARTIAL SEIZURES	19	9%
4	STATUS EPILEPTICUS	5	2%
5	COMPLEX PARTIAL SEIZURES	3	1%
6	EPILEPSIA PARTIALIS CONTINUA	2	1%

FIGURE 3: DISTRIBUTION OF TYPE OF SEIZURES



Among the new onset seizures studied most were generalized tonic clonic seizures (71%), followed by partial seizures with secondary generalization (15%).

TABLE 5: DISTRIBUTION OF SEIZURE TYPES IN VARIOUS AGE GROUPS

S.NO	TYPE OF SEIZURE	AGE IN YEARS	
		MEAN	SD
1	GENERALISED TONIC CLONIC SEIZURES	36.16	17.510
2	PARTIAL SEIZURES WITH SECONDARY GENERALISATION	49.17	19.320
3	SIMPLE PARTIAL SEIZURES	46.84	18.750
4	STATUS EPILEPTICUS	37.20	7.200
5	COMPLEX PARTIAL SEIZURES	47.33	19.500
6	EPILEPSIA PARTIALIS CONTINUA	67.50	14.840

FIGURE 4: DISTRIBUTION OF SEIZURE TYPES IN VARIOUS AGE GROUPS

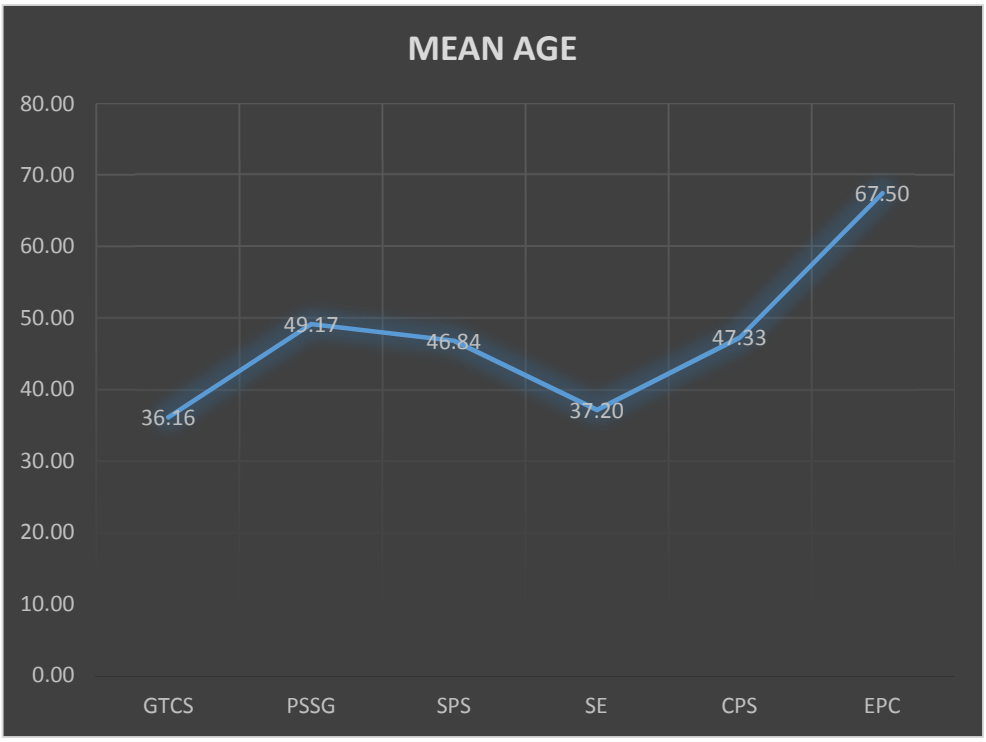
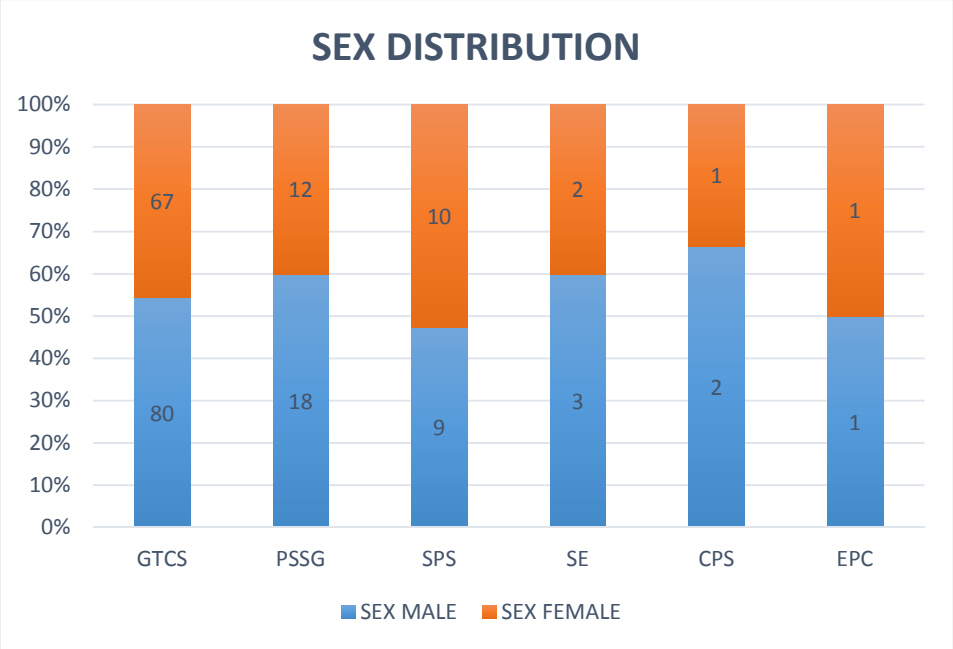


TABLE 6: DISTRIBUTION OF SEIZURE TYPES IN MALES & FEMALES

S.NO	TYPE OF SEIZURE	SEX	
		MALE	FEMALE
1	GENERALISED TONIC CLONIC SEIZURES	80	67
2	PARTIAL SEIZURES WITH SECONDARY GENERALISATION	18	12
3	SIMPLE PARTIAL SEIZURES	9	10
4	STATUS EPILEPTICUS	3	2
5	COMPLEX PARTIAL SEIZURES	2	1
6	EPILEPSIA PARTIALIS CONTINUA	1	1
	TOTAL	113	93

FIGURE 5: DISTRIBUTION OF SEIZURE TYPES IN MALES & FEMALES



All seizure types were more common in males except simple partial seizures which were slightly more common in females.

TABLE 7: ASSOSCIATION OF SYMPTOMS WITH SEIZURES

S.NO	SYMPTOMS	NO OF PATIENTS	PERCENTAGE
1	FEVER	50	24%
2	HEADACHE	83	40%
3	VOMITING	78	37%

FIGURE 6: ASSOSCIATION OF SYMPTOMS WITH SEIZURES

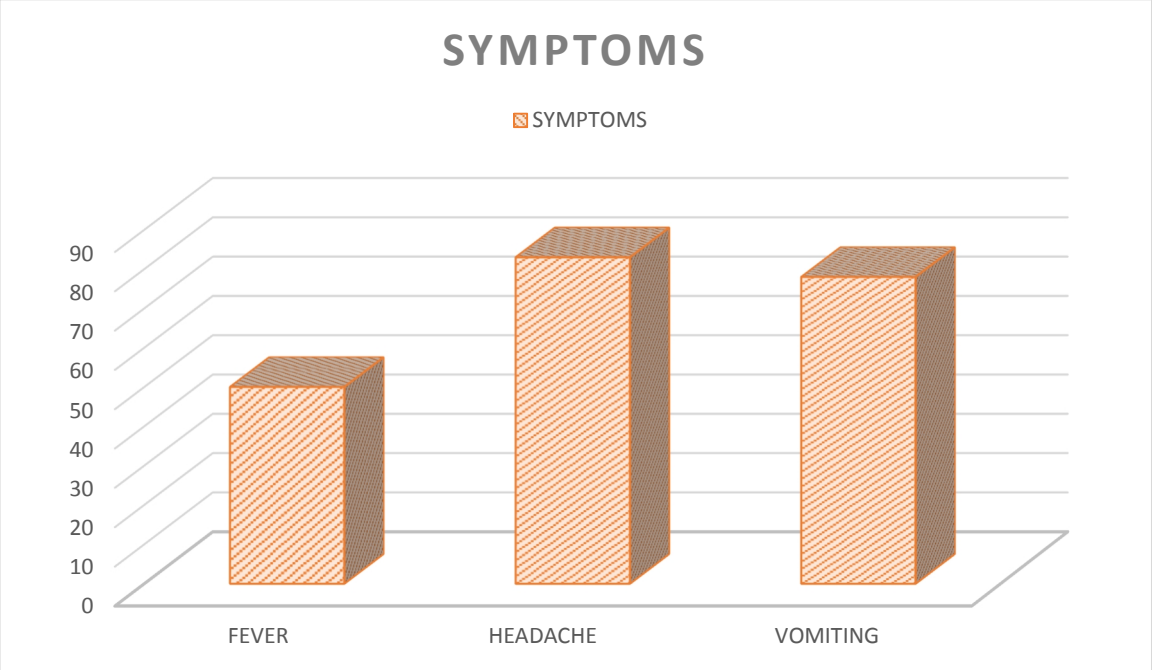
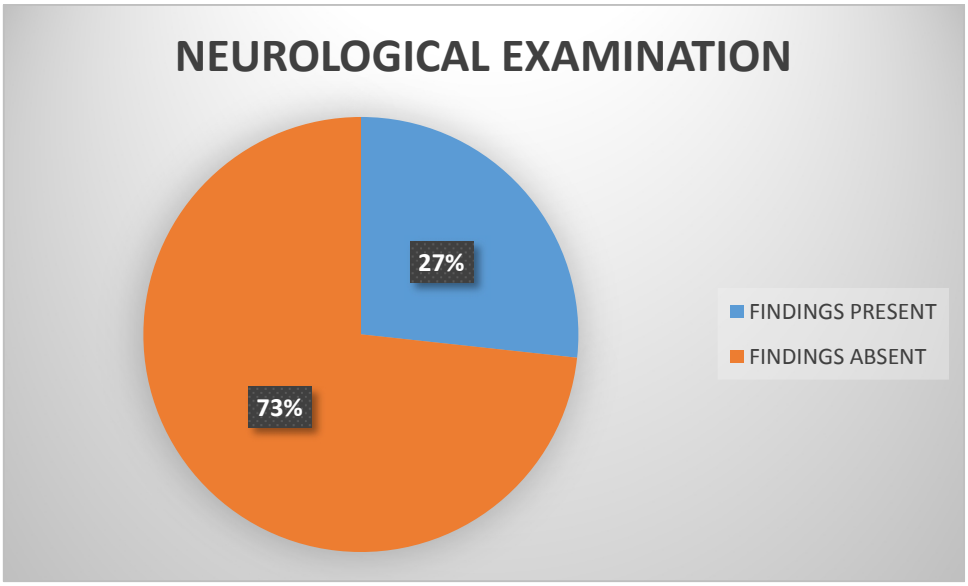


TABLE 8: DISTRIBUTION OF NEUROLOGICAL FINDINGS IN SEIZURES

S.NO	NEUROLOGICAL EXAMINATION	NO OF PATIENTS	PERCENTAGE
1	FINDINGS PRESENT	55	27%
2	FINDINGS ABSENT	151	73%

FIGURE 7: DISTRIBUTION OF NEUROLOGICAL FINDINGS IN SEIZURES

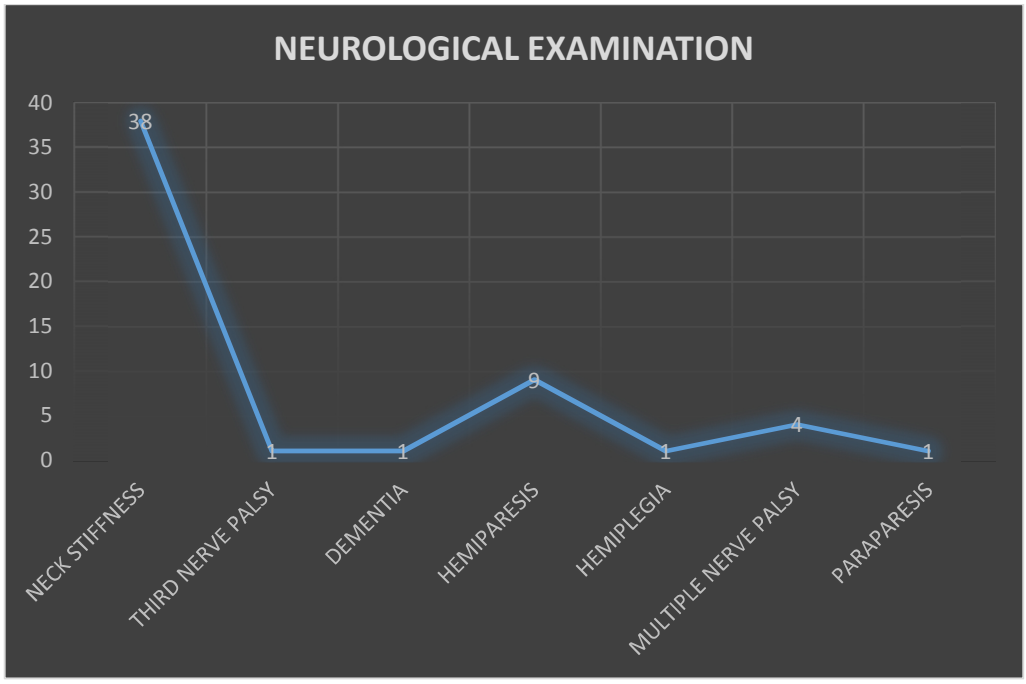


Among the patients studied, only 27% had findings on neurological examination.

TABLE 9: ASSOCIATED NEUROLOGICAL FINDINGS IN SEIZURES

S.NO	NEUROLOGICAL EXAMINATION	NO OF PATIENTS	PERCENTAGE
1	NECK STIFFNESS	38	69%
2	THIRD NERVE PALSY	1	2%
3	DEMENTIA	1	2%
4	HEMIPARESIS	9	16%
5	HEMIPLEGIA	1	2%
6	MULTIPLE NERVE PALSY	4	7%
7	PARAPARESIS	1	2%

FIGURE 8: ASSOCIATED NEUROLOGICAL FINDINGS IN SEIZURES



The most common clinical finding was neck stiffness, found in 69% of the cases. Hemiparesis was the most common focal neurological deficit, seen in 16% of patients.

TABLE 10: PATTERN OF DISTRIBUTION OF NECK STIFFNESS IN VARIOUS ETIOLOGIES

S.NO	NECK STIFFNESS	NO OF PATIENTS	PERCENTAGE
1	CNS INFECTION	35	92%
2	SUB ARACHNOID HEMORRHAGE	3	8%
	TOTAL	38	100%

FIGURE 9: PATTERN OF DISTRIBUTION OF NECK STIFFNESS IN VARIOUS ETIOLOGIES

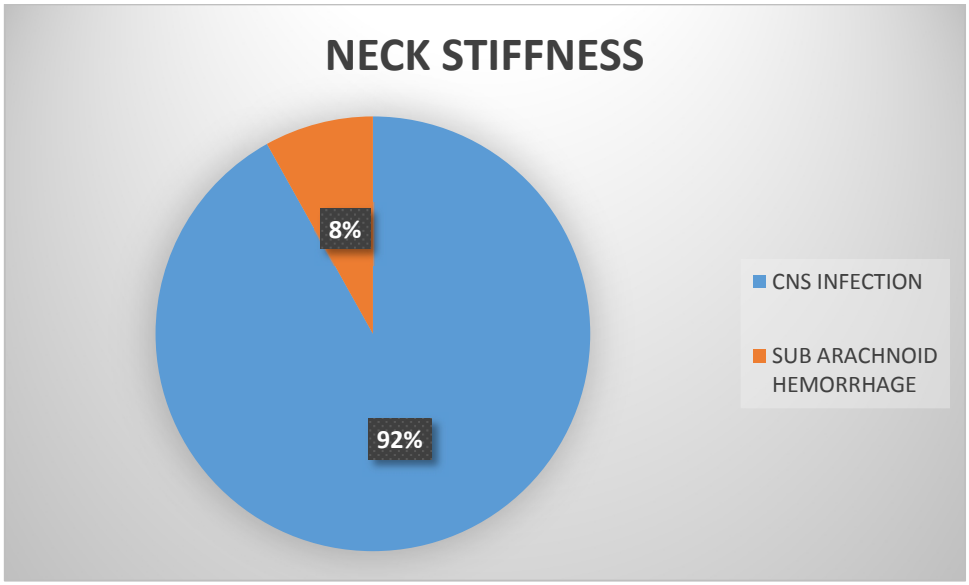


TABLE 11: DISTRIBUTION OF VARIOUS AETIOLOGIES

S.NO	ETIOLOGY	NO OF PATIENTS	PERCENTAGE
1	CNS INFECTION	48	23%
2	CEREBROVASCULAR ACCIDENT	55	27%
3	METABOLIC CAUSE	27	13%
4	TUMOR	11	5%
5	POISONING	6	3%
6	IDIOPATHIC	21	10%
7	ALCOHOL WITHDRAWAL	13	6%
8	ECLAMPSIA	14	7%
9	FAMILIAL ADULT MYOCLONIC EPILEPSY	10	5%
10	POST DIALYSIS	1	1%
	TOTAL	206	100%

Among the cases of new onset seizures studied 27% were caused by cerebrovascular accidents which included ischemic stroke, haemorrhagic stroke, etc. Next common cause was neurological infections like TB meningitis, viral encephalitis. Together these two account for 50% of the cases. Among the aetiologies post dialysis seizures and poisoning accounted for lowest number of cases.

FIGURE 10: DISTRIBUTION OF VARIOUS AETIOLOGIES

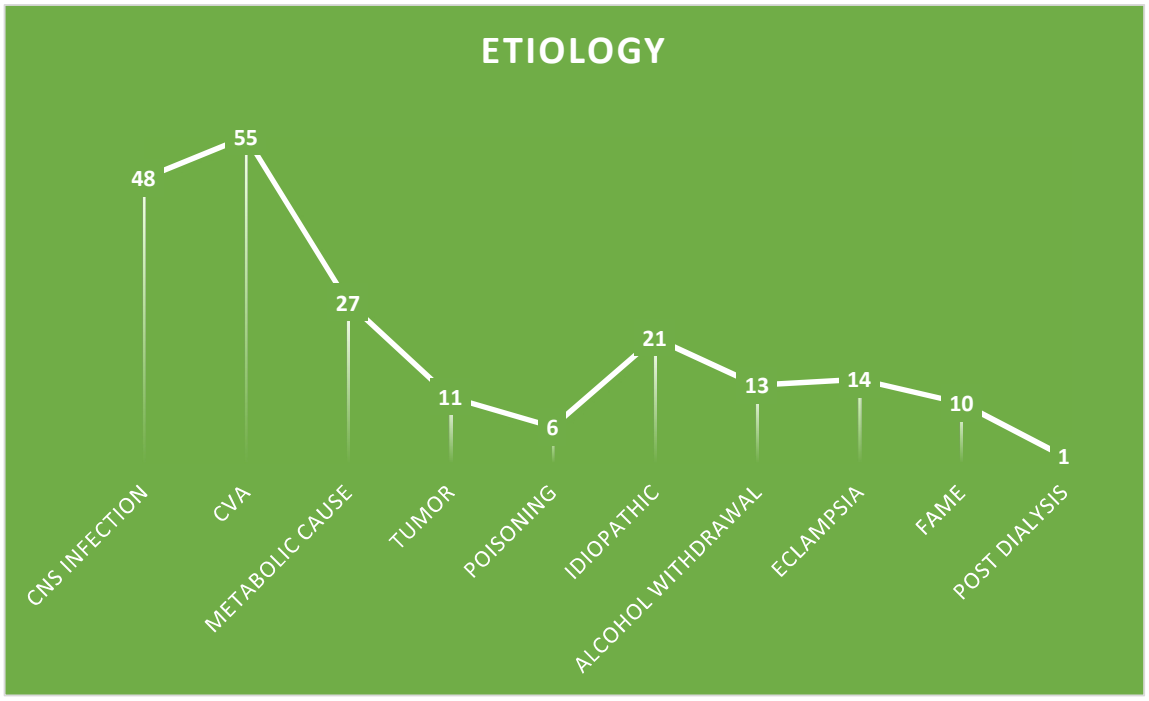


TABLE 12: ASSOCIATION OF VARIOUS TYPES OF CEREBRO VASCULAR ACCIDENTS WITH NEW ONSET SEIZURES

S.NO	CEREBROVASCULAR ACCIDENTS	NO OF PATIENTS	PERCENTAGE
1	ISCHEMIC STROKE	27	50%
2	HEMORRHAGIC STROKE	12	21%
3	CEREBRAL VENOUS THROMBOSIS	12	21%
4	SUB ARACHNOID HAEMORRHAGE	3	6%
5	SUB DURAL HEMORRHAGE	1	2%
	TOTAL	55	100%

FIGURE 11: ASSOCIATION OF VARIOUS TYPES OF CEREBRO VASCULAR ACCIDENTS WITH NEW ONSET SEIZURES

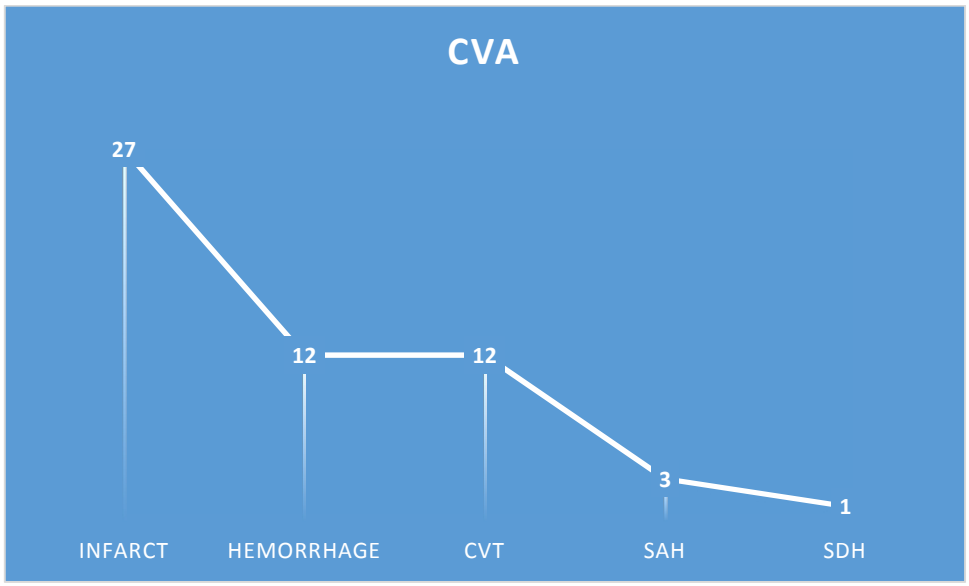
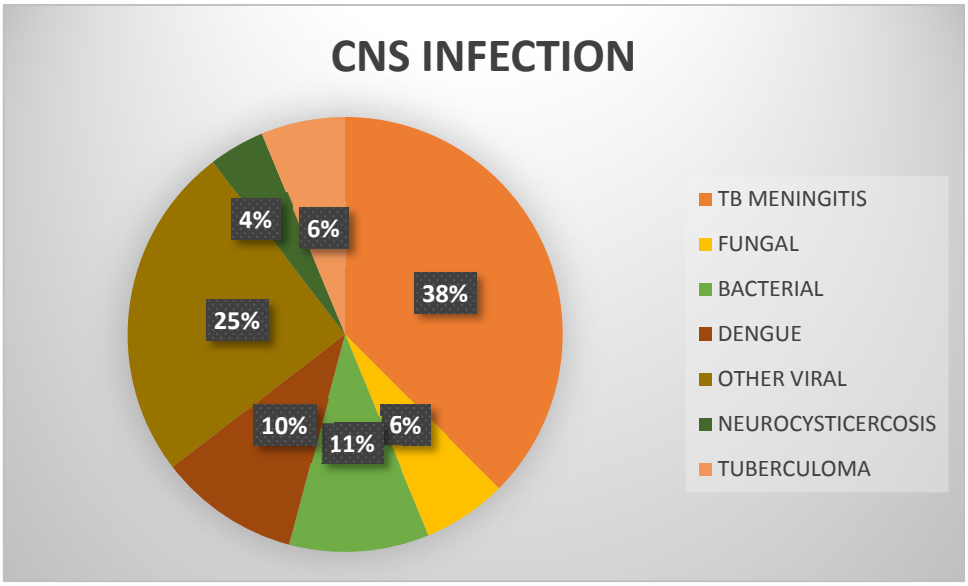


TABLE 13: DISTRIBUTION OF CNS INFECTION IN NEW ONSET SEIZURES

S.NO	CNS INFECTION	NO OF PATIENTS	PERCENTAGE
1	TB MENINGITIS	18	38%
2	FUNGAL	3	6%
3	BACTERIAL	5	11%
4	DENGUE	5	10%
5	OTHER VIRAL	12	25%
6	NEUROCYSTICERCOSIS	2	4%
7	TUBERCULOMA	3	6%
	TOTAL	48	100%

FIGURE 12: DISTRIBUTION OF CNS INFECTION IN NEW ONSET SEIZURES

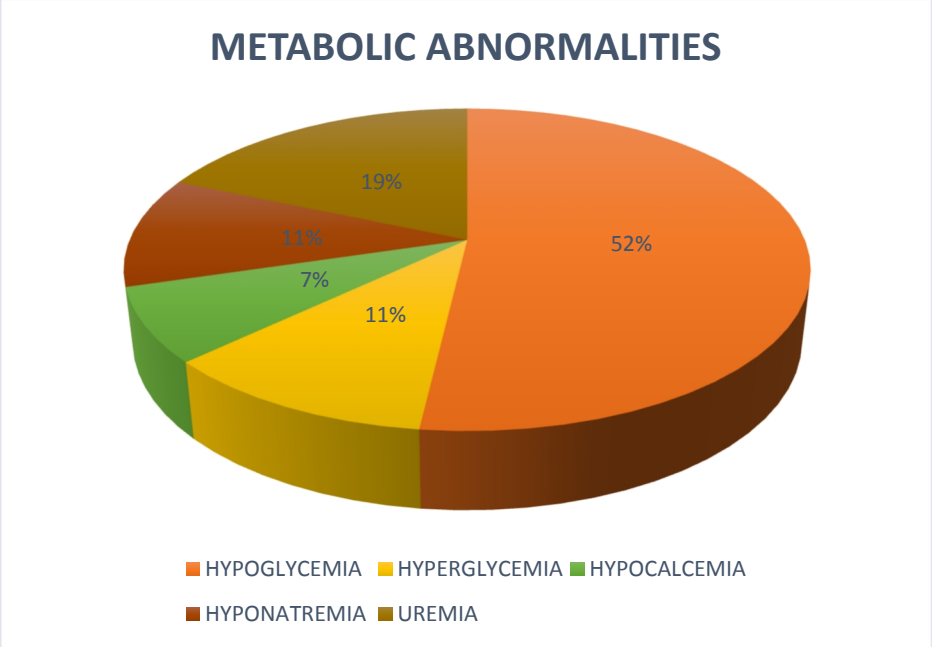


Tuberculous meningitis was the leading cause of new onset seizures with infective aetiology. This was followed by viral infections like dengue and herpes simplex of which the former accounted for approximately 30% of the cases. Rarer causes were fungal infections, neurocysticercosis and tuberculoma. 2 cases of mucormycosis were noted in elderly diabetic patients and 1 case of cryptococcal meningitis in a HIV patient.

TABLE 14: TYPES OF METABOLIC ABNORMALITIES

S.NO	METABOLIC ABNORMALITIES	NO OF PATIENTS	PERCENTAGE
1	HYPOGLYCEMIA	14	52%
2	HYPERGLYCEMIA	3	11%
3	HYPOCALCEMIA	2	7%
4	HYPONATREMIA	3	11%
5	UREMIA	5	19%
	TOTAL	27	100%

FIGURE 13: TYPES OF METABOLIC ABNORMALITIES



More than half of metabolic seizures were due to hypoglycaemia. Second most common aetiology for metabolic seizure was uremia. Electrolyte abnormalities

contributed to 18% of the metabolic seizures and included hyponatremia and hypocalcemia.

TABLE 15: PATTERN OF TUMOUR

S.NO	TUMOUR	NO OF PATIENTS	PERCENTAGE
1	PRIMARY	7	64%
2	SECONDARY	4	36%
	TOTAL	11	100%

FIGURE 14: PATTERN OF TUMOUR

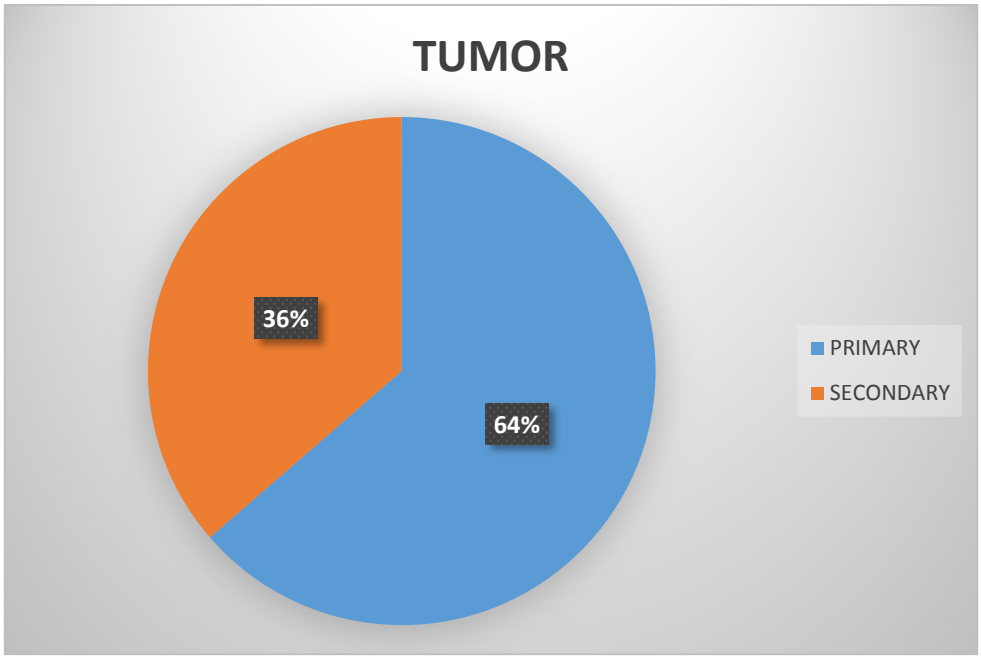
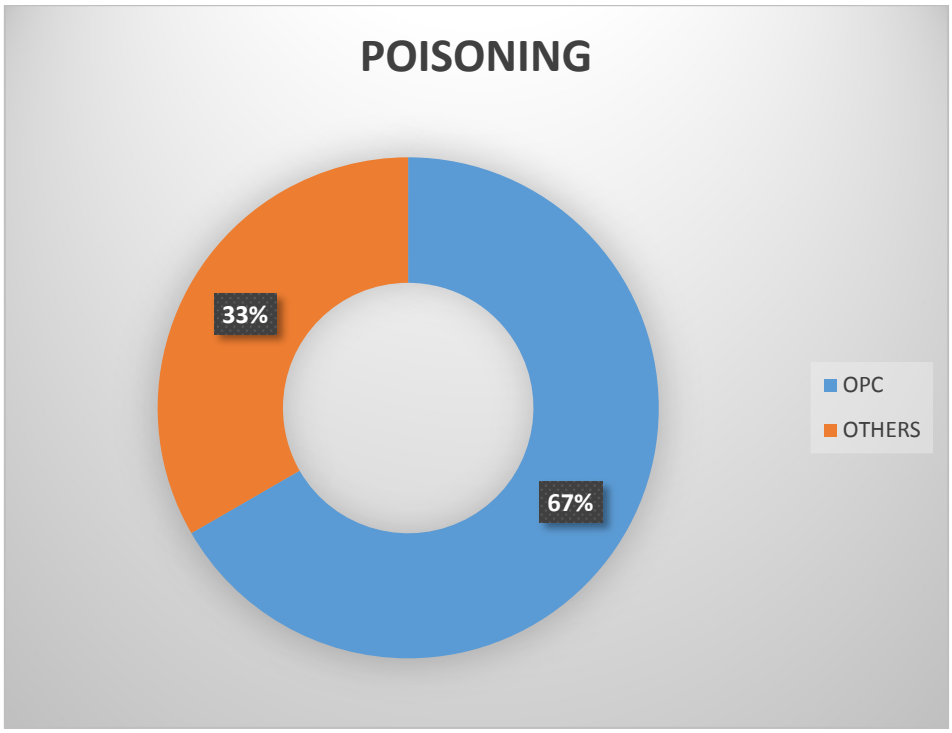


TABLE 16: TYPES OF POISOINING CAUSING NEW ONSET SEIZURES

S.NO	POISONING	NO OF PATIENTS	PERCENTAGE
1	ORGANOPHOSPHOROUS COMPOUNDS	4	67%
2	OTHERS	2	33%
	TOTAL	6	100%

FIGURE 15: TYPES OF POISOINING CAUSING NEW ONSET SEIZURES



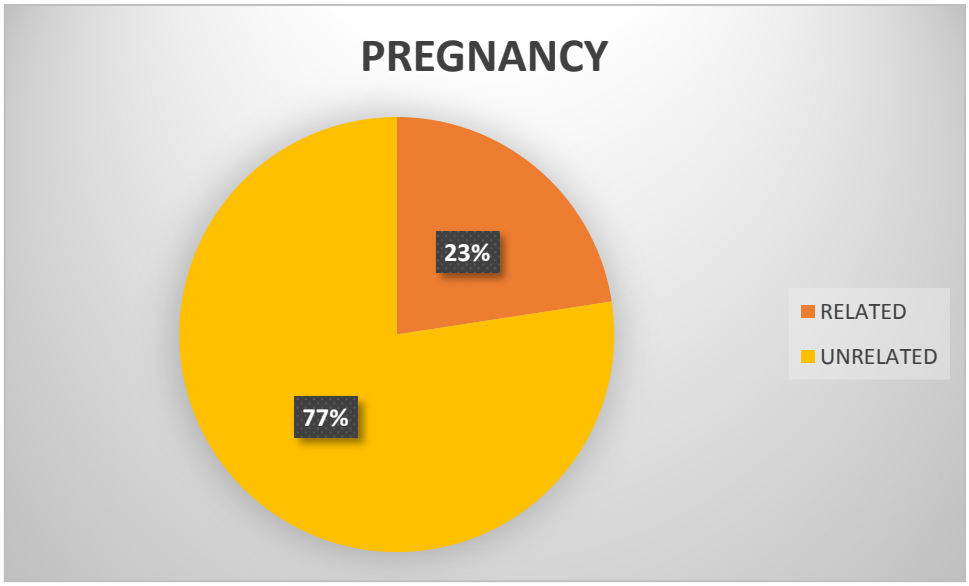
Organophosphorous compounds accounted for maximum number of poison induced seizures in this region due to easy accessibility to the farmer population

in the form of pesticide. Other causes for poisoning included camphor poisoning and eucalyptus oil consumption.

TABLE17: ASSOCIATION WITH PREGNANCY

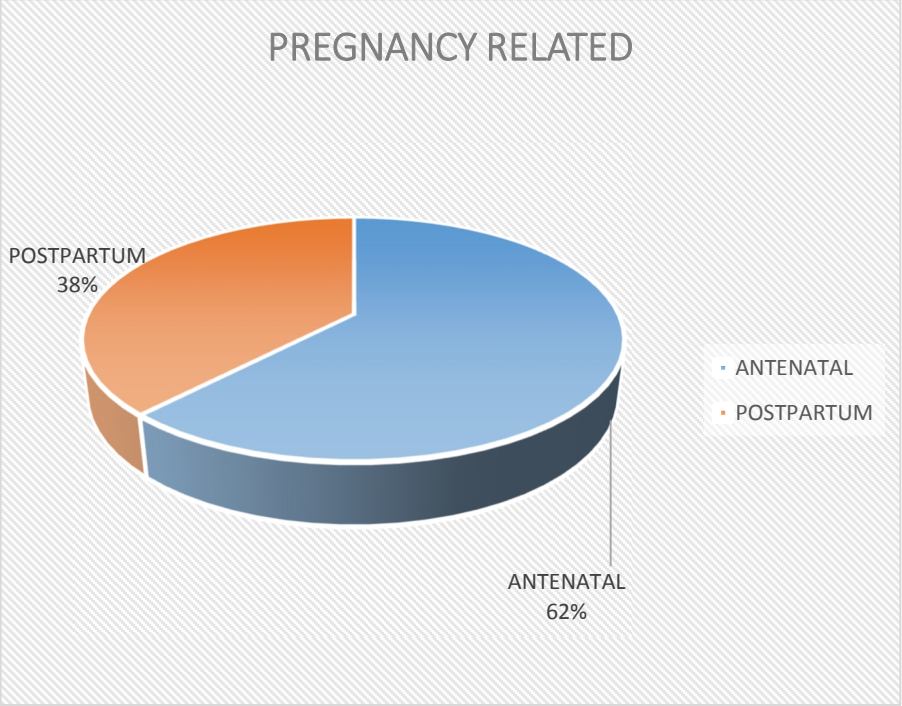
S.NO	PREGNANCY	NO OF PATIENTS	PERCENTAGE
1	RELATED	21	23%
2	UNRELATED	72	77%
	TOTAL	93	100%

FIGURE 16: ASSOCIATION WITH PREGNANCY



Among the 93 females enrolled in the study 23% (n=21) of cases were related to pregnancy. Of these 13 cases were antenatal and 8 cases were post-partum.

FIGURE 17: TEMPORAL RELATION WITH PREGNANCY

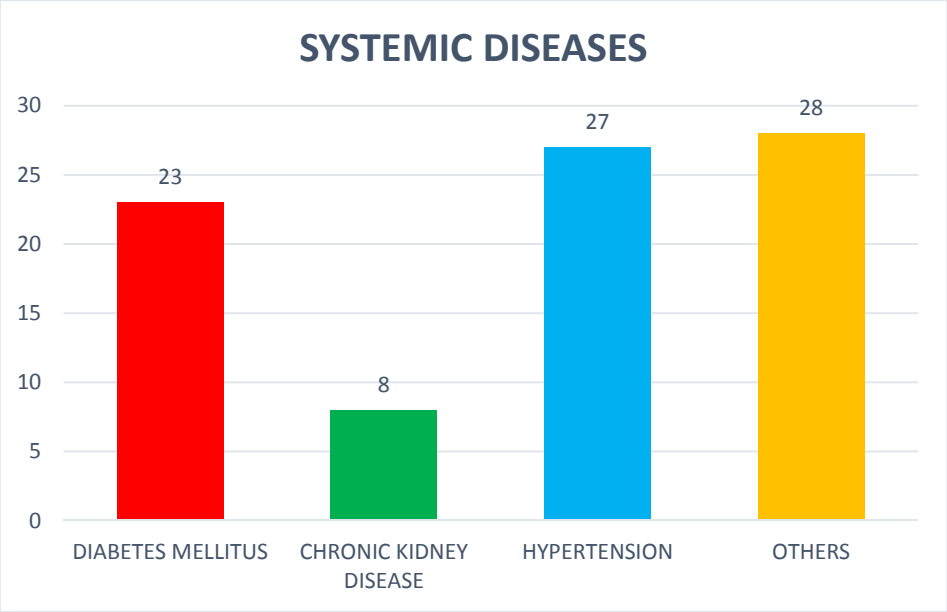


Pregnancy related causes for new onset seizures were eclampsia and cerebral venous thrombosis. Cerebral venous thrombosis was common in postpartum women and eclampsia related seizures were common in antenatal women.

TABLE 18: CO-EXISTING SYSTEMIC DISEASES

S.NO	MEDICAL ILLNESS	NO OF PATIENTS	PERCENTAGE
1	DIABETES MELLITUS	23	11%
2	CHRONIC KIDNEY DISEASE	8	4%
3	HYPERTENSION	27	13%
4	OTHERS	28	14%
	TOTAL	86	42%

FIGURE 18: CO-EXISTING SYSTEMIC DISEASES



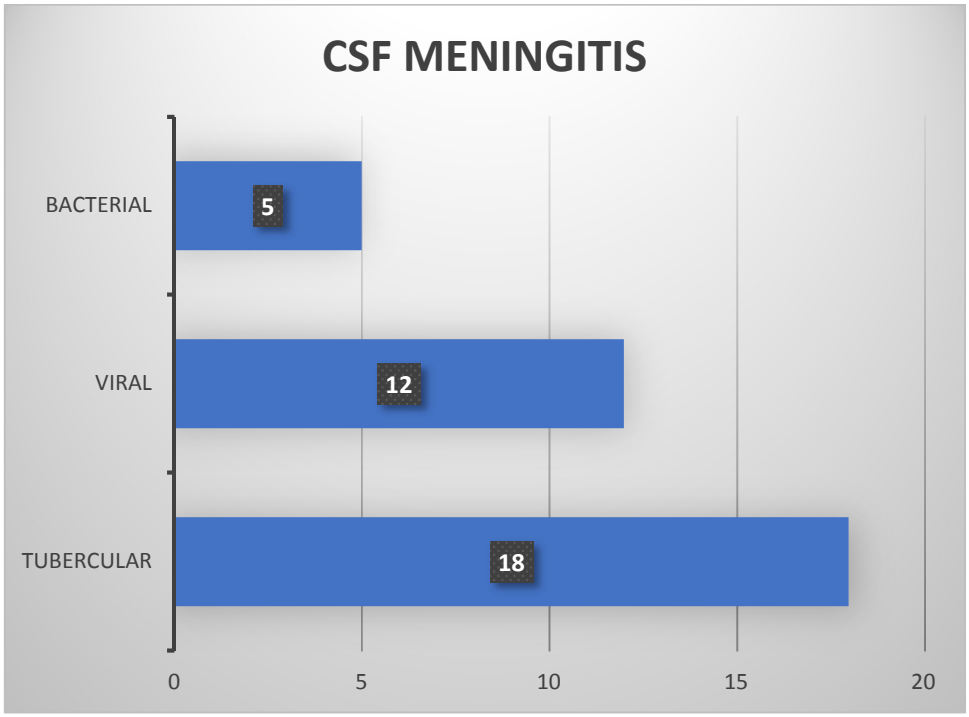
42% of the study population had coexisting systemic diseases, most common of which were hypertension and diabetes. Others included pulmonary tuberculosis, systemic lupus erythematosus, malignancies, etc.

Cerebrospinal fluid analysis was done for suspected cases of neurological infections, when not contraindicated and most common picture was of tuberculous type.

TABLE 19: VARIOUS CSF ANALYSIS PATTERN

S.NO	CSF MENINGITIS	NO OF PATIENTS	PERCENTAGE
1	TUBERCULAR	18	52%
2	VIRAL	12	34%
3	BACTERIAL	5	14%
	TOTAL	35	100%

FIGURE 19: VARIOUS CSF ANALYSIS PATTERN

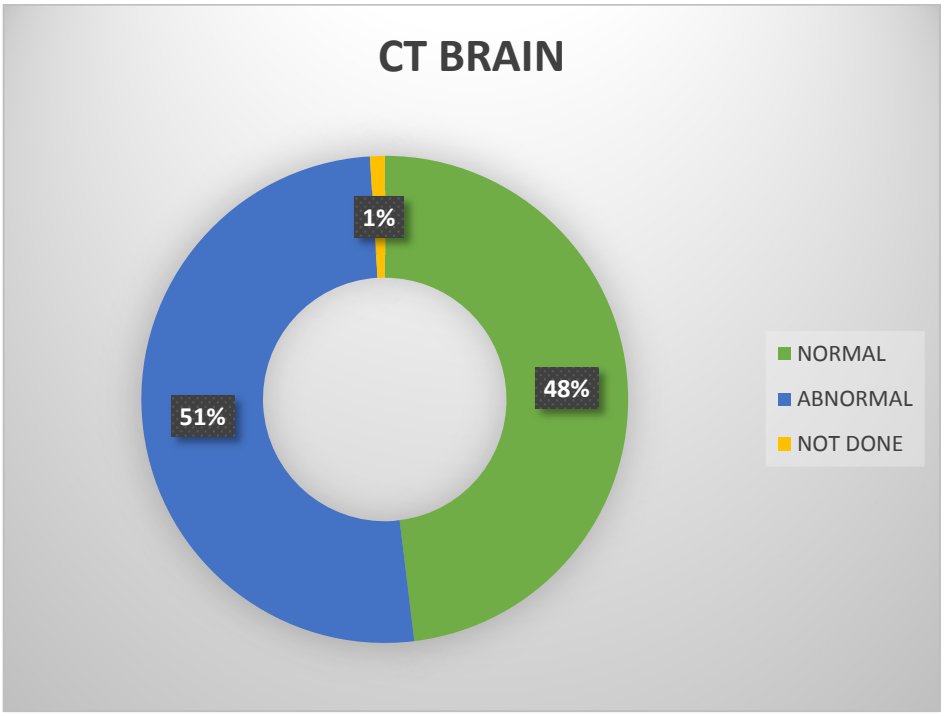


CT Brain was done for all cases except 1 antenatal case for which MRI Brain was done to confirm the diagnosis.

TABLE 20: CT BRAIN

S.NO	CT BRAIN	NO OF PATIENTS	PERCENTAGE
1	NORMAL	99	48%
2	ABNORMAL	105	51%
3	NOT DONE	2	1%
	TOTAL	206	100%

FIGURE 20: CT BRAIN



CT Brain was abnormal in 51% of the cases

TABLE 21: CT FINDINGS AMONG THE PARTICIPANTS

CT FINDINGS	NO OF PATIENTS	PERCENTAGE
INFARCT	18	17%
CALCIFIED GRANULOMA	2	2%
GLIOTIC CHANGES	8	8%
CEREBRAL EDEMA	18	17%
CEREBRALVENOUS THROMBOSIS	9	8%
SPACE OCCUPYING LESION	6	6%
HEMORRHAGE	17	16%
OTHERS	27	26%

FIGURE 21: CT FINDINGS AMONG THE PARTICIPANTS

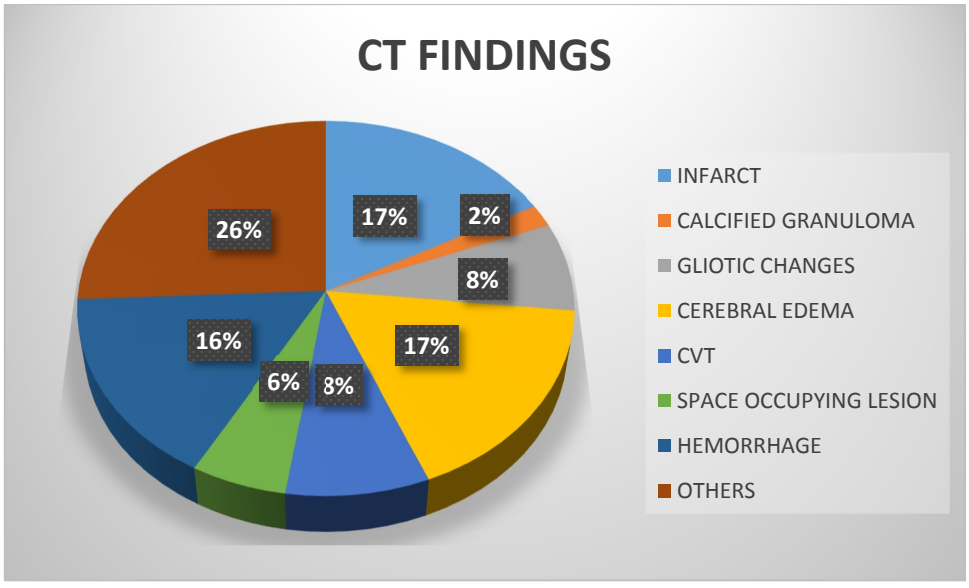


TABLE 22: CT FINDINGS VS TYPES OF SEIZURES

S.NO	CT FINDINGS	TYPE OF SEIZURE					
		GTCS	PSSG	SPSS	SE	CPS	EPC
1	INFARCT	14	3	2	0	1	0
2	CALCIFIED GRANULOMA	0	1	0	1	0	0
3	GLIOTIC CHANGES	0	5	3	0	0	0
4	CEREBRAL EDEMA	16	0	1	0	1	0
5	CEREBRAL VENOUS THROMBOSIS	5	4	0	0	0	0
6	SPACE OCCUPYING LESION	1	3	2	0	0	0
7	HEMORRHAGE	7	5	5	0	0	0
8	OTHERS	6	5	2	0	0	1
	TOTAL	49	26	15	1	2	1

In patients with generalised tonic clonic seizures, most common findings were cerebral oedema and infarct. Among patients who had partial seizures with secondary generalisation most common CT brain abnormality was gliotic changes. Epilepsia partialis continua was noticed in 2 cases of metabolic seizures.

Fundus examination was done in all the cases enrolled for the study and was found abnormal in 47 patients out of 206.

TABLE 23: FUNDUS EXAMINATION

S.NO	FUNDUS	NO OF PATIENTS	PERCENTAGE
1	NORMAL	159	77%
2	ABNORMAL	47	23%
	TOTAL	206	100%

FIGURE 22: FUNDUS EXAMINATION

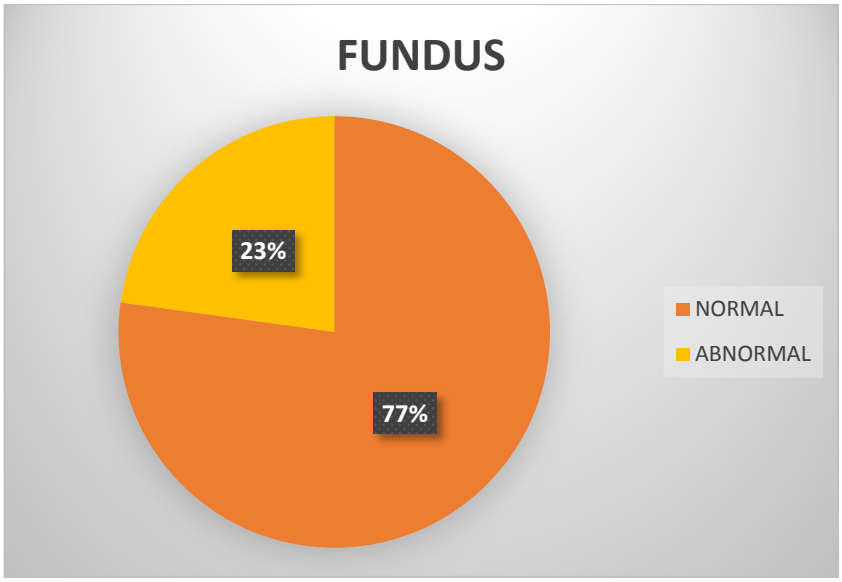
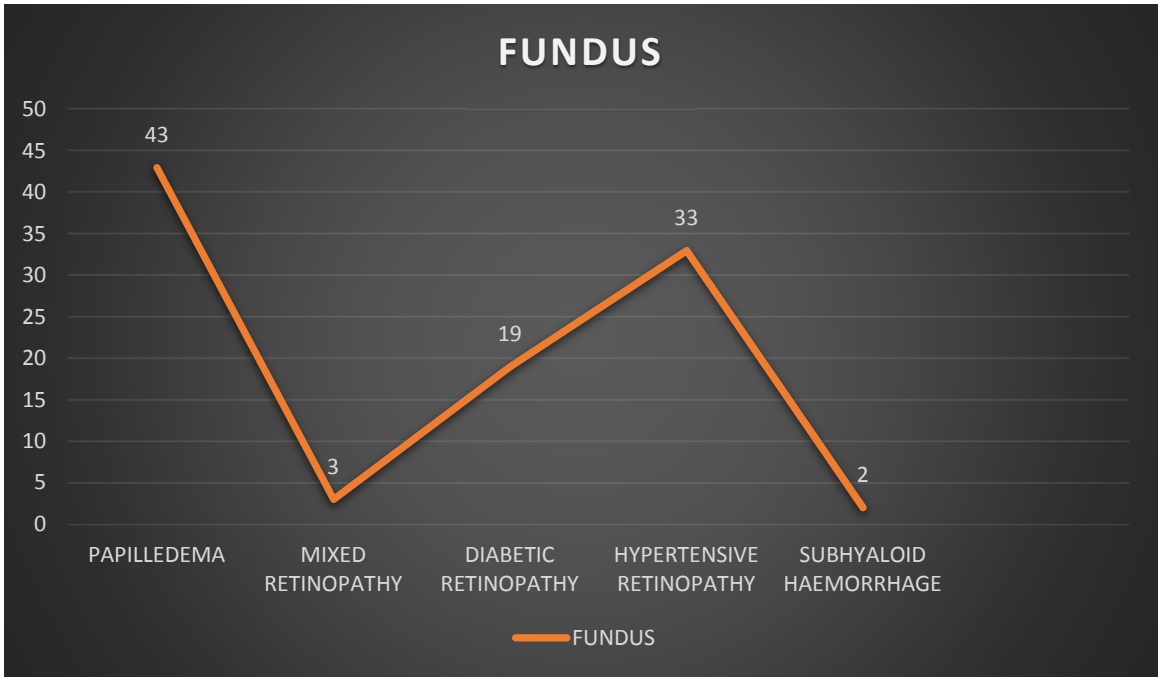


TABLE 24: FUNDUS CHANGES OBSERVED IN THE STUDY GROUP

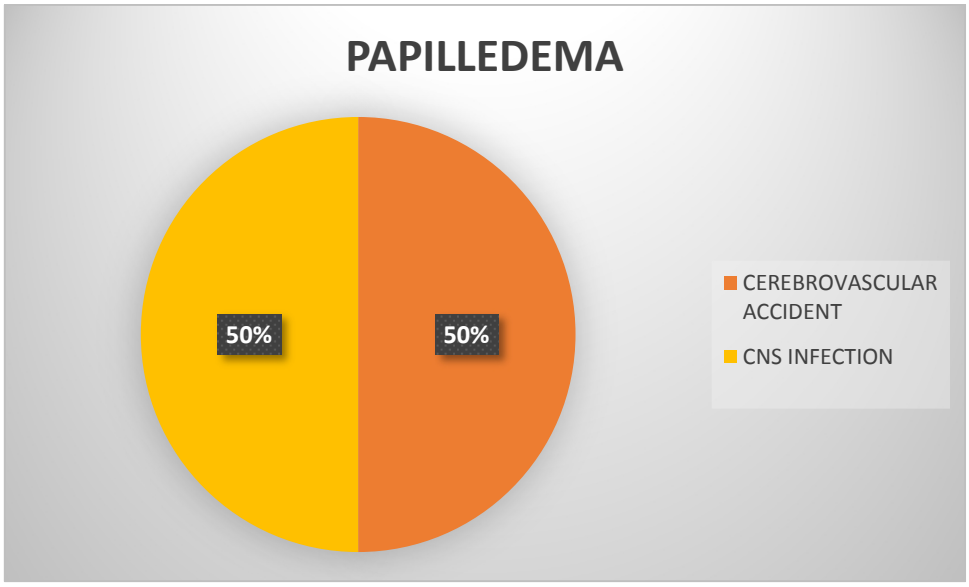
S.NO	FUNDUS	NO OF PATIENTS	PERCENTAGE
1	PAPILLEDEMA	20	43%
2	MIXED RETINOPATHY	2	3%
3	DIABETIC RETINOPATHY	9	19%
4	HYPERTENSIVE RETINOPATHY	15	33%
5	SUB HYALOID HEMORRHAGE	1	2%
	TOTAL	206	100%

FIGURE 23: FUNDUS CHANGES OBSERVED IN THE STUDY GROUP



Papilledema was the most common fundus abnormality noted in 43% of the cases.

FIGURE 24: DISTRIBUTION OF PAPILLEDEMA IN VARIOUS CONDITIONS

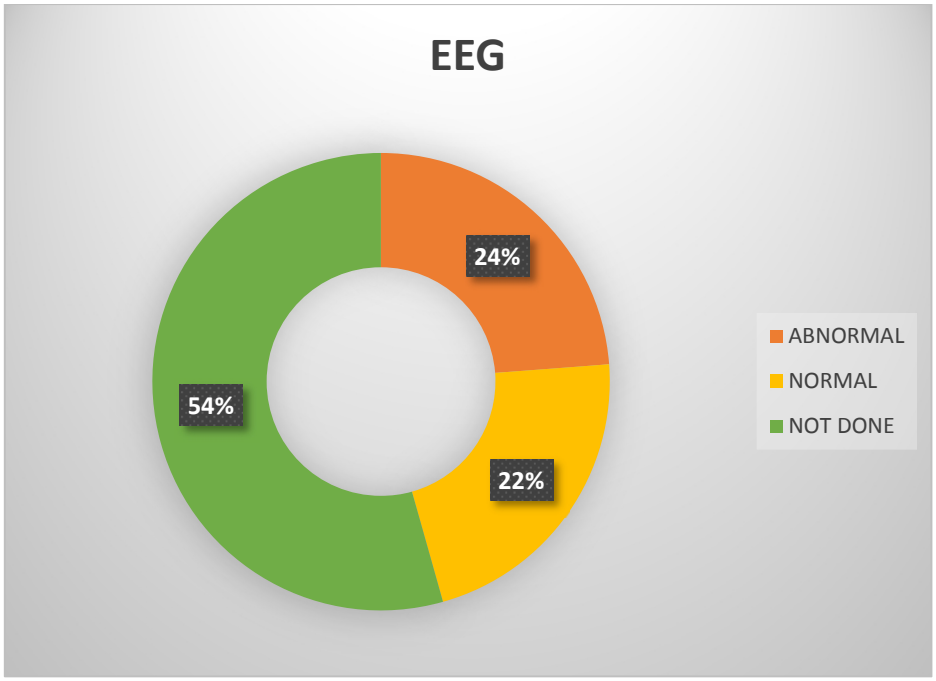


Papilledema was observed in 10 patients with cerebrovascular accidents and 10 patients with CNS infections.

TABLE 25: ELECTROENCEPHALOGRAM

S.NO	EEG	NO OF PATIENTS	PERCENTAGE
1	ABNORMAL	49	24%
2	NORMAL	45	22%
	NOT DONE	112	54%

FIGURE 25: ELECTROENCEPHALOGRAM

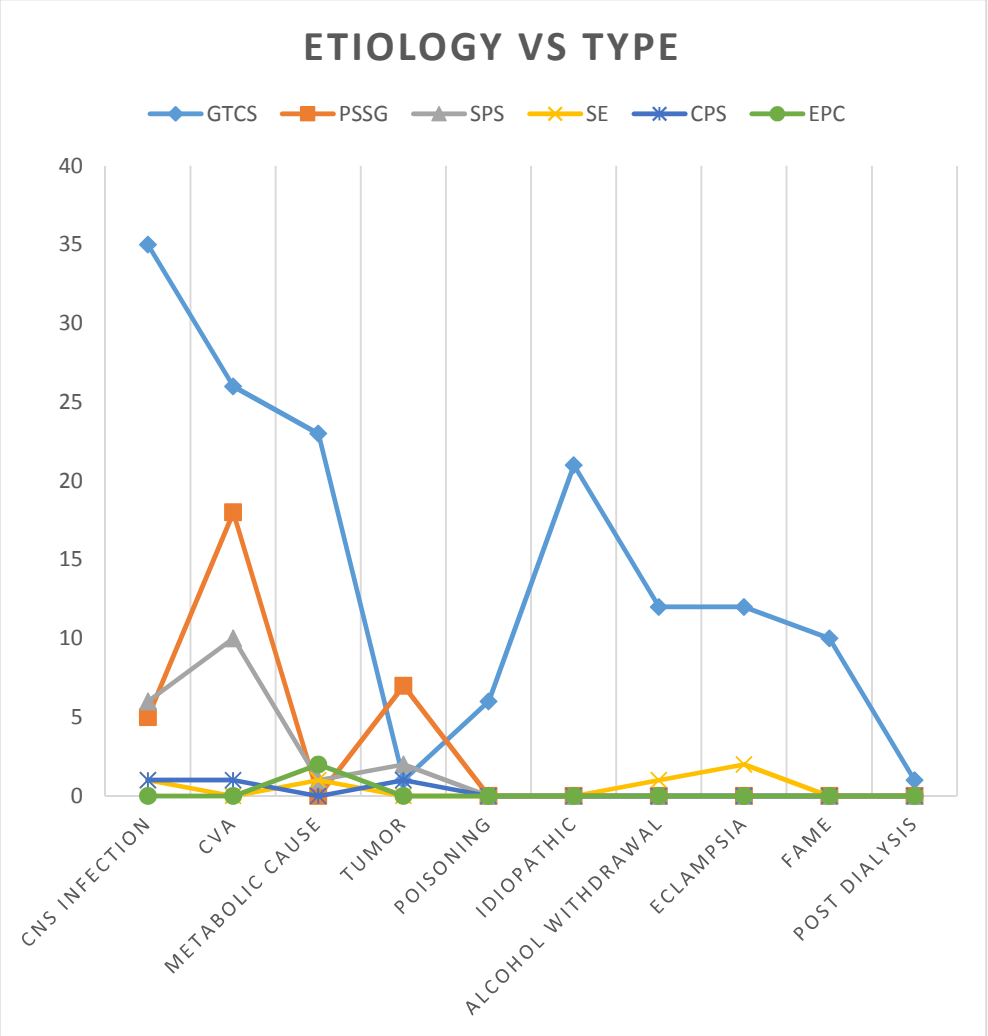


EEG was done for 46% of cases and was found to be abnormal in 52% of them. Most common abnormality was bilateral epileptiform activity. Other abnormalities noted were bilateral sharp synchronous waves, sharp spike and wave patterns & slowing of wave forms.

TABLE 26: DISTRIBUTION OF AETIOLOGY VS TYPE

S.NO	ETIOLOGY	TYPE OF SEIZURE					
		GTCS	PSSG	SPS	SE	CPS	EPC
1	CNS INFECTION	35	5	6	1	1	0
2	CEREBROVASCULAR ACCIDENT	26	18	10	0	1	0
3	METABOLIC CAUSE	23	0	1	1	0	2
4	TUMOR	1	7	2	0	1	0
5	POISONING	6	0	0	0	0	0
6	IDIOPATHIC	21	0	0	0	0	0
7	ALCOHOL WITHDRAWAL	12	0	0	1	0	0
8	ECLAMPSIA	12	0	0	2	0	0
9	FAMILIAL ADULT MYOCLONIC EPILEPSY	10	0	0	0	0	0
10	POST DIALYSIS	1	0	0	0	0	0
	TOTAL	147	30	19	5	3	2

FIGURE 26: DISTRIBUTION OF AETIOLOGY VS TYPE



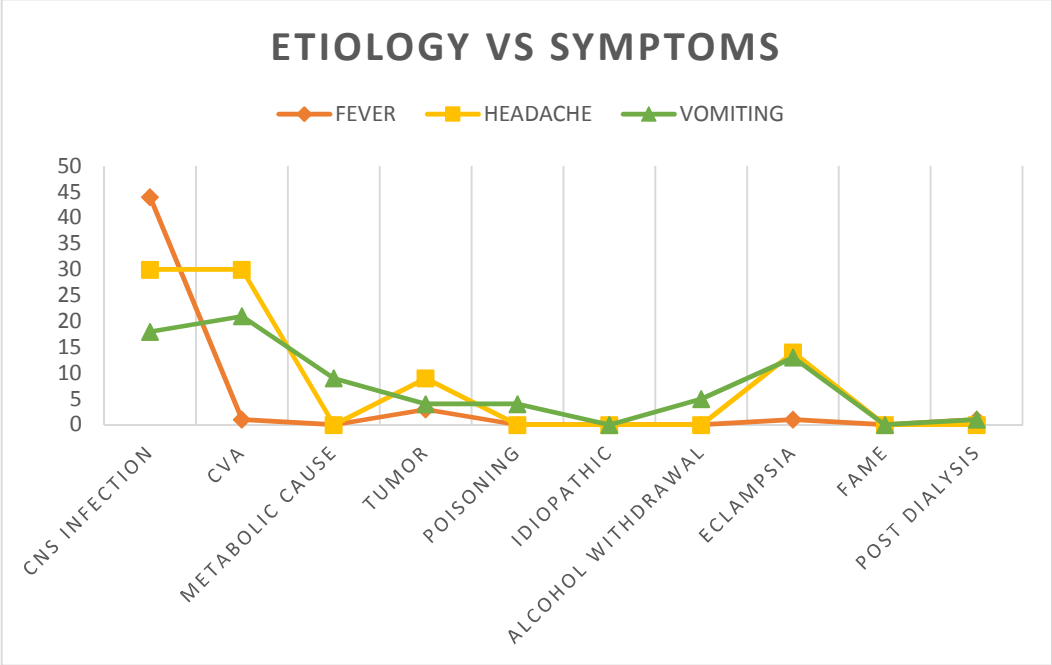
Generalised tonic clonic seizures was the most common seizure type in all aetiologies discussed except in cases tumours when partial seizures with secondary generalisation outnumbered other types.

TABLE 27: PATTERN OF DISTRIBUTION OF SYMPTOMS IN VARIOUS ETIOLOGIES

S.NO	ETIOLOGY	FEVER	HEAD ACHE	VOMITING
1	CNS INFECTION	44	30	18
2	CEREBROVASCULAR ACCIDENT	1	30	21
3	METABOLIC CAUSE	0	0	9
4	TUMOR	3	9	4
5	POISONING	0	0	4
6	IDIOPATHIC	0	0	0
7	ALCOHOL WITHDRAWAL	0	0	5
8	ECLAMPSIA	1	14	13
9	FAMILIAL ADULT MYOCLONIC EPILEPSY	0	0	0
10	POST DIALYSIS	1	0	1
	TOTAL	40	83	75

FIGURE 27: PATTERN OF DISTRIBUTION O SYMPTOMS

IN VARIOUS ETIOLOGIES



Headache was the most common symptom in the study population followed by vomiting. In cases with an infective aetiology fever was almost always present.

TABLE 28: PATTERN OF DISTRIBUTION OF SYMPTOMS & SIGNS IN CNS INFECTIONS

S.NO	CNS INFECTION	FEVER	HEAD ACHE	VOMITING	NECK STIFFNESS
1	TB MENINGITIS	16	13	8	17
2	FUNGAL	3	3	2	1
3	BACTERIAL	5	2	1	5
4	DENGUE	5	4	3	3
5	OTHER VIRAL	12	5	4	8
6	NEUROCYSTICERCOSIS	1	1	0	0
7	TUBERCULOMA	2	2	0	0
	TOTAL	44	30	18	34

TABLE 29: PATTERN OF DISTRIBUTION OF SYMPTOMS & SIGNS IN CEREBROVASCULAR ACCIDENTS

S.NO	CVA	FEVER	HEADACHE	VOMITING	NECK STIFFNESS
1	INFARCT	1	8	3	0
2	HEMORRHAGE	0	5	4	0
3	CVT	0	12	11	0
4	SAH	0	3	2	3
5	SDH	0	1	0	0
	TOTAL	1	29	20	3

FIGURE 28: PATTERN OF DISTRIBUTION OF SYMPTOMS & SIGNS IN CEREBROVASCULAR ACCIDENTS

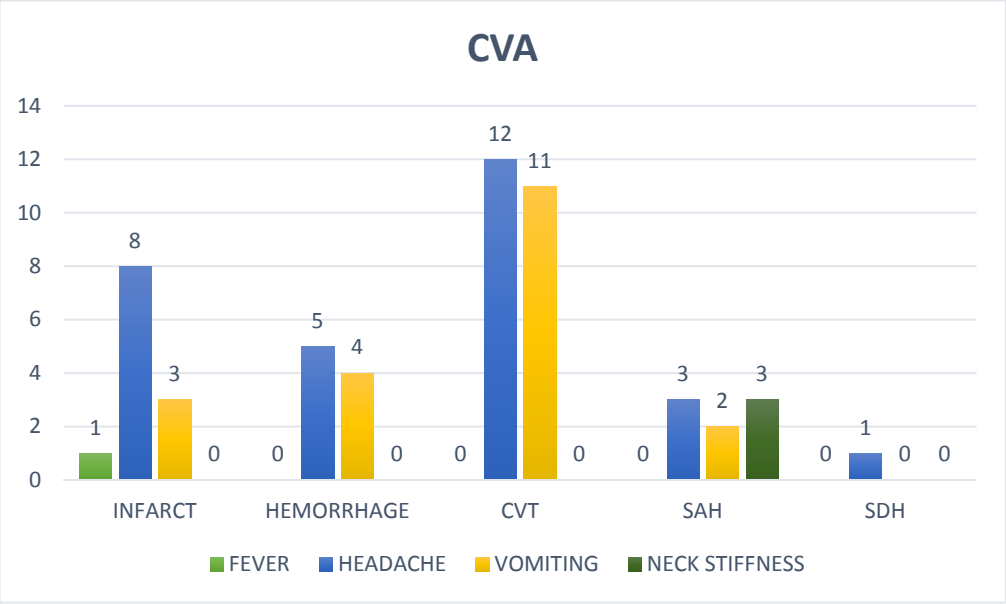


TABLE 30: ETIOLOGY VS AGE GROUPS

S.NO	AGE GROUP	INFECTIONS	CVA	METABOLIC	IDIOPATHIC	TUMOUR	ALCOHOL WITHDRAWAL	POISONING	ECLAMPSIA	FAME	POST DIALYSIS	TOTAL
1	13-20	17	2	2	8	1	0	2	1	2	0	35
2	21-30	15	6	2	8	3	4	2	6	3	0	49
3	31-40	7	5	4	3	1	6	1	6	3	1	37
4	41-50	4	11	3	2	3	3	1	1	1	0	29
5	51-60	2	14	5	0	1	0	0	0	1	0	23
6	61-70	2	10	7	0	2	0	0	0	0	0	21
7	71-80	1	7	2	0	0	0	0	0	0	0	10
8	>80	0	0	2	0	0	0	0	0	0	0	2
	TOTAL	48	55	27	21	11	13	6	14	10	1	206

Among the age group 12-30 years maximum cases had an infective aetiology followed by idiopathic. TB meningitis was the foremost cause amongst infective aetiologies, next was viral meningitis. Dengue related neurological complications were noted only in young age. Cerebrovascular accidents accounted for almost 50% cases in the age group 41-80 years.

Cerebral venous thrombosis was the principal aetiology attributed for cerebrovascular accidents in young age where as ischemic stroke was the leading cause for the same in the elderly population. Metabolic causes had maximum contribution in age group 51-70 years. Primary brain tumors were common in young age while secondaries attributed to major chunk in elderly. Alcohol withdrawal seizures were seen in the age group of 21-50 years. Familial adult myoclonic epilepsy & idiopathic generalised epilepsy as a cause for new onset seizures were pre-eminent in younger age group.

Female predominance in young age cerebrovascular accidents was noteworthy and was attributed to pregnancy and cerebral venous thrombosis. 75% cases of cerebral venous thrombosis was noted in females and 78% of them were pregnant ladies. In age group of 41-60 years males outnumbered females in causing cerebrovascular accident and related new onset seizures.

TABLE 31: AGE & SEX DISTRIBUTION OF COMMON ETIOLOGIES

S.NO	AGE GROUP	CNS INFECTIONS		CEREBROVASCULAR ACCIDENT		METABOLIC	
		MALE	FEMALE	MALE	FEMALE	MALE	FEMALE
1	13-20	8	9	0	2	1	1
2	21-30	10	5	0	6	0	2
3	31-40	3	4	3	2	2	2
4	41-50	1	3	9	2	2	1
5	51-60	2	0	11	3	1	4
6	61-70	1	1	3	7	5	2
7	71-80	1	0	6	1	2	0
8	>80	0	0	0	0	2	0
	TOTAL	26	22	32	23	16	11

DISCUSSION

About 5 – 10% of population develop at least one episode of seizure during their lifetime. There are more than 50 million people suffering from epilepsy worldwide, majority of whom reside in developing countries. Several studies have shown that infectious causes are the leading etiology of new onset seizures in developing countries like India. Lack of education and awareness, access to healthcare services, cultural taboos and superstitions all account for the wide treatment gap between diagnosis and management of the same. An accurate description of the etiological distribution of seizures can help in early management of the target population and strengthen epilepsy services in those underserved areas. Here, in our study at a tertiary care centre in south Tamil Nadu, India, we are trying to illustrate the spectrum of etiologies commonly encountered.

In our study, the age group of 12- 50 years accounted for more than 70% of the cases. This was in confirmation with another etiological study of epilepsy done by Satishchandra et al[24] in south India, where the same age group constituted approximately two thirds of the cases both in urban and rural areas. In a recent similar study from north India[40], more than 75% of the cases were in this age group. Our study had a sex prevalence in favour of males over females in the ratio of 1.2:1, similar to other studies.

We found that the most common type of seizure was GTCS, in more than two third of the cases, which was similar to another study from a different region in south India[41], where they recorded GTCS in 57%of the patients. GTCS was found in the mean age of 36 years with a standard deviation of 17. 54% of the

GTCS cases were male patients. Similarly, males accounted for 60% of the partial seizures with secondary generalisation. Only group with female predominance was simple partial seizures (53%).

Among the clinical features, headache was present in 40% of the cases in our study akin to other studies[41]. Neck stiffness was not a common finding, but was highly prevalent among cases of infective aetiology and sub arachnoid haemorrhage.

S.NO	ETIOLOGY	SUDHIR et al,2015	QURAISHI et al,2015	ASHWIN et al,2017	OUR STUDY
1	CNS INFECTION	39.70%	38%	17%	23%
2	CEREBROVASCULAR ACCIDENT	26.50%	30%	21%	27%
3	METABOLIC CAUSE	15.3%	2%	15%	13%
4	TUMOR	3%	2%	-	5%
5	ALCOHOL WITHDRAWAL	-	8%	5%	6%
6	IDIOPATHIC	-	20%	18%	10%
7	OTHERS	-	-	16%	16%

As per the study conducted, maximum number of new onset seizures were attributable to cerebrovascular accidents (27%) parallel with other studies by Quraishi et al[42], Sudhir et al[43] which revealed a similar incidence despite being second most common cause behind CNS infection in their studies. CNS infections constituted 23% of the cases unlike the older studies, but analogous to the study

by Ashwin et al[41]. Metabolic seizures were ascribed an incidence of 13% -15% in all studies except Quraishi et al which showed very minimal contribution (2%).

On further analysis of the subgroups under cerebrovascular accident, ischemic stroke accounted for half of the cases followed by cerebral venous thrombosis which accounted for 22% in our study in coherence with all the aforementioned studies. Among neurological infections TB meningitis was the most common cause in our study while two studies showed neurocysticercosis as the most common cause.

Most of the CNS infection patients presented with GTCS (~80%), followed by SPS& PSSG which was similar to study conducted by Quraishi et al. Cerebrovascular accident was the most common cause for PSSG accounting for 60% followed by tumours and infective causes. Metabolic seizures were mainly GTCS in present study as was discussed by Sudhir et al. Alike to Quraishi et al alcohol withdrawal seizures were primarly of GTCS type.

Cerebral venous thrombosis showed a female to male ratio of 3:1 as was described by Coutinho et al[44]. Familial adult onset myoclonic epilepsy which was never mentioned in other similar studies accounted for 5% of cases as a cause for new onset seizures. This entity was unique to a community residing in this part of south Tamilnadu as mentioned by Mahadevan et al in a landmark study conducted in the same region which was one among the first studies to talk on these cases outside Far East Asia[45].

CONCLUSION

Following are the conclusions derived from "STUDY ON ETIOLOGICAL PROFILE OF NEW ONSET SEIZURES IN ADULTS IN A TERTIARY CARE CENTRE".

- New onset seizures had a male predominance with male: female ratio of 1.2:1.
- First episode of seizure are most common in 21-30 years with incidence of 24%
- Overall most common seizure type was generalised tonic clonic seizures, with incidence 71%.
- Most common aetiology was cerebrovascular accidents (27%) followed by CNS infections (23%).
- Among cerebrovascular accidents most common was ischemic stroke (50%) while TB meningitis attributed to maximum cases among infections (38%).
- Cerebral venous thrombosis accounted for highest number of cases among cerebrovascular accidents in younger age group with a male to female ratio 3:1.
- Familial adult onset myoclonic epilepsy accounted for 5%, not mentioned as a cause for new onset seizures in similar studies and was unique to a community in this part of south Tamilnadu.

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PROFORMA

NAME: AGE: SEX:

ADDRESS:

PRESENT ILLNESS

Associated with:

FEVER: HEADACHE: VOMITING:

AURAS: POST ICTAL CONFUSION:

OTHERS:

TYPE OF SEIZURES:

PRECIPITATING FACTORS:

ALCOHOL WITHDRAWAL HISTORY: PRE ECLAMPSIA:

SIGNIFICANT PAST HISTORY

DM: HTN: PTB: CVA: HIV: OTHERS:

FAMILY HISTORY:

PERSONAL HISTORY:

ALCOHOL INTAKE: SMOKING:

TOXINS/DRUGS: ANTENATAL/POSTPARTUM:

VITALS

PR: BP: PULSE: TEMPERATURE:

EXAMINATION

Signs of meningeal irritation:

Cranial nerves:

Motor system:

Sensory system:

Cerebellum:

Others:

INVESTIGATIONS

RBS: UREA: CREATININE:

TC: DC: PLATELET COUNT: ESR:

Na^+: K^+: Ca^{2+}: CSF ANALYSIS:

CT BRAIN: CONTRAST CT: MRI BRAIN:

EEG: FUNDUS:

FINAL DIAGNOSIS

ABBREVIATIONS

CA:	CARCINOMA
CECT:	CONTRAST ENHANCED COMPUTED TOMOGRAPHY
CKD:	CHRONIC KIDNEY DISEASE
CPS:	COMPLEX PARTIAL SEIZURES
CSF:	CEREBROSPINAL FLUID
CT:	COMPUTED TOMOGRAPHY
CVA:	CEREBRO VASCULAR ACCIDENT
CVT:	CEREBRAL VENOUS THROMBOSIS
DM:	DIABETES MELLITUS
EEG:	ELECTROENCEPHALOGRAM
EPC:	EPILEPSIA PARTIALIS CONYINUA
F:	FEMALE
FAME:	FAMILIAL ADULT MYOCLONIC EPILEPSY
GTCS:	GENERALISED TONIC CLONIC SEIZURES
HIV:	HUMAN IMMUNODEFICIENCY VIRUS
HTN:	HYPERTENSION
IGE:	IDIOPATHIC GENERALISED EPILEPSY

M:	MALE
MEN (B):	BACTERIAL MENINGITIS
MEN (T):	TUBERCULOUS MENINGITIS
MEN (V):	VIRAL MENINGITIS
MRI:	MAGNETIC RESONANCE IMAGING
N:	NORMAL
NEURO E:	NEUROLOGICAL EXAMINATION
NCC:	NEUROCYSTICERCOSIS
PSSG:	PARTIAL SEIZURES WITH SECONDARY GENERALISATION
PTB:	PULMONARY TUBERCULOSIS
RBS:	RANDOM BLOOD SUGAR
SAH:	SUB ARACHNOID HEMORRHAGE
SDH:	SUB DURAL HEMORRHAGE
SE:	STATUS EPILEPTICUS
SLE:	SYSTEMIC LUPUS ERYTHEMATOSUS
SPS:	SIMPLE PARTIAL SEIZURES
+	PRESENT:
-:	ABSENT

S. no	Name	Age	Sex	Medical illness	FEVER	HEADACHE	VOMITING	NEURO E	RBS	urea	Na	Ca	platelet	CT brain/CECT	CSF	FUNDUS	EEG finding	DIAGNOSIS	TYPE
1	DEV HARISH	15	M	PTB	+	+	-	Neck stiffness	115	36	136	9	1.9	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
2	KALIDAS	20	M		+	+	+	Neck stiffness	123	30	134	8.6	1.48	CEREBRAL EDEMA	MEN(V)	N	NOT DONE	VIRAL MENINGITIS	GTCS
3	MADAKANNU	68	F	HTN /DM/OLD CVA	+	-	-	hemiparesis	306	46	130	9.2	2.2	INFARCT		MR	N	CVA-ischemic	PSSG
4	MAHARAJAN	45	M	HTN/DM	-	-	-		**40**	28	128	9.5	2.5	N		DR	N	HYPOGLYCEMIC	GTCS
5	DHARSHINI	17	F		+	+	-	Neck stiffness	94	35	132	9	4	CEREBRAL EDEMA	MEN(B)	N	N	BACTERIAL MENINGITIS	GTCS
6	MADHUBALA	19	F		-	-	-		110	28	130	8.8	2.9	N		N	ABNORMAL	IGE	GTCS
7	PETCHIAMMAL	30	F	CKD	-	-	+		103	193	134	9	1.5	N		N	NOT DONE	UREMIC SEIZURES	GTCS
8	NATHIYA	30	F	POSTPARTUM	-	+	-		100	18	136	9	2.9	CVT		PAPILLEDEMA	NOT DONE	CVA-CVT	PSSG
9	KUTTIAMMAL	70	F	CA LUNG	-	+	-	hemiparesis	102	22	132	9.7	3	BRAIN SECONDARIES		N	NOT DONE	SECONDARIES BRAIN/CA LUNG	PSSG
10	VELMURUGAN	35	M		-	-	-		80	25	132	9.8	2.7	N		N	N	ALCOHOL WITHDRAWAL SEIZURES	GTCS
11	SARASWATHY	30	F	POSTPARTUM	-	+	+	paraparesis	98	32	138	8.8	4	CVT		PAPILLEDEMA	NOT DONE	CVA-CVT	GTCS
12	TAMILARASI	20	F		-	-	+		87	35	135	9	3.5	SOL		N	ABNORMAL	TUMOUR	SPS
13	MUNIYANDI	46	M		-	-	+		98	34	133	10	4.5	N		N	N	OPC POISONING	GTCS
14	SELVAMATHA	35	F	ANTENATAL	-	+	-		136	28	137	9.8	3.4	N		N	NOT DONE	ECLAMPSIA	GTCS
15	ARUMUGAM	79	M	DM	-	-	-	DEMENTIA	187	34	136	8.5	2.8	GLIOTIC CHANGES		N	ABNORMAL	CVA-ISCHEMIC	SPS
16	KARUPAYEE	70	F	DM	-	-	-		**38**	76	135	9	3.5	CEREBRAL ATROPHY		DR	NOT DONE	HYPOGLYCEMIC SEIZURES	GTCS
17	DARSHITHA	50	F		-	-	-		92	40	133	9	2.5	N		N	ABNORMAL	IGE	GTCS
18	RAMKUMAR	24	M		-	+	-		110	18	134	9.2	3.4	SOL		N	ABNORMAL	TUMOUR	GTCS
19	GANESAN	61	M	HTN	-	-	-	hemiparesis	118	36	136	9	1.7	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	GTCS
20	UTCHIMAHALI	70	M	DM	-	-	+		145	53	**110**	9.4	1.8	N		DR	NOT DONE	HYPONATREMIA	GTCS
21	GOVINDAN	59	M	HTN	-	+	-	Neck stiffness	105	42	127	9.3	2.1	SAH		SHH	NOT DONE	SUB ARACHNOID HEMORRHAGE	GTCS
22	SUNDAR	25	M	PTB	+	+	+	Neck stiffness	123	22	140	9	1.9	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	N	NOT DONE	TB MENINGITIS	SPS
23	VASANTHA	20	F		-	-	-		78	30	133	9.2	3.4	N		N	ABNORMAL	FAME	GTCS
24	KALAI	55	M	HTN	-	-	-	hemiparesis	108	38	129	9.3	2.7	INFARCT		N	NORMAL	CVA-ISCHEMIC	GTCS
25	MUTHAVALAN	16	M		-	-	-		87	18	135	9.1	3.1	N		N	ABNORMAL	IGE	GTCS
26	MARIA ANTHONY	57	F	DM	-	-	-		**448**	52	134	8.6	2.73	N		DR	NOT DONE	HYPERGLYCEMIC SEIZURES	EPC
27	PRIYA	43	F		-	-	-	hemiparesis	116	35	140	9.2	2	RING ENHANCING LESION		N	NOT DONE	TUBERCULOMA	SPS
28	LOKKA	40	M		-	-	-		120	32	134	10.2	1.95	CALCIFIED GRANULOMA		N	NOT DONE	NCC	PSSG
29	SEETHARAMAN	23	M		-	-	-		132	45	129	9.7	2.5	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	GTCS
30	RAMYA	14	F		+	+	-	Neck stiffness	96	85	130	8.8	3.5	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
31	POOMARI	49	F		-	-	-		112	18	133	9.2	4.5	N		N	ABNORMAL	IGE	GTCS
32	PETCHI	35	F		-	+	+		110	48	135	9.5	2.9	N		N	NORMAL	ECLAMPSIA	GTCS
33	MARIAMMAL	44	F		-	-	-	hemiparesis	220	54	132	9	3.1	HEMORRHAGE		N	NOT DONE	CVA-HEMORRHAGIC	SPS
34	NATARAJAN	80	M	OLD CVA	-	-	-	hemiparesis	126	45	137	9.5	3.5	GLIOTIC CHANGES		N	ABNORMAL	CVA-ISCHEMIC	PSSG
35	PARVATHY	70	F	HTN	-	-	-	HEMIPLEGIA	110	34	136	9	3.5	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	GTCS

36	THIRUPATHI	17	M	T1DM	-	-	-		**48**	40	144	9.3	3.6	N		N	NOT DONE	HYPOGLYCEMIC SEIZURES	GTCS
37	LATHEEF	40	M		-	+	+		96	33	142	9.5	4	CVT		PAPILLEDEMA	NOT DONE	CVA-CVT	PSSG
38	KALYAN	72	M		-	-	-	hemiparesis	119	39	135	8	4.2	INFARCT		N	N	CVA-ISCHEMIC	SPS
39	SIVASAKTHI	24	F	ANTENATAL	-	+	+		124	44	132	9.2	4.5	N		N	NOT DONE	ECLAMPSIA	GTCS
40	RASATHI	35	F		-	-	-		117	26	143	9.5	4.8	N		N	NOT DONE	OPC POISONING	GTCS
41	JEGANATHAN	60	M	HTN	-	-	-	hemiparesis	93	42	137	9	2.5	N		HR	ABNORMAL	CVA-ISCHEMIC	GTCS
42	MURUGESHWARI	27	F		-	-	-		120	18	139	10.1	3.2	N		N	ABNORMAL	FAME	GTCS
43	PITCHAMMAL	25	F		-	-	-		207	24	136	9.5	3.4	N		N	ABNORMAL	IGE	GTCS
44	NAGARAJAN	39	M	HTN	-	+	-	3rd N palsy	115	32	136	9.5	3.2	SDH		N	NOT DONE	SDH	PSSG
45	RAJENDRAN	13	M		+	-	-	Neck stiffness	122	35	134	10	4.2	N	MEN(V)	N	NOT DONE	VIRAL MENINGITIS	GTCS
46	VALLITHAI	68	F	HTN	-	+	+	Neck stiffness	136	36	141	9.7	3.2	SAH		PAPILLEDEMA	NOT DONE	SUB ARACHNOID HEMORRHAGE	PSSG
47	KULANTHAI VELU	45	M	HTN	-	-	-		140	45	136	9.2	3.3	INFARCT		HR	ABNORMAL	CVA-ISCHEMIC	GTCS
48	VALLIAMMAL	36	F		-	-	-		100	38	137	**5.8**	4.1	N		N	NOT DONE	HYPOCALCEMIA	SPS
49	MURUGESAN	52	M		-	+	-		112	36	132	9.2	2.5	INFARCT		N	ABNORMAL	CVA-ISCHEMIC	GTCS
50	PARAMESHWARI	24	F		-	-	-		129	22	145	9.6	3.2	N		N	ABNORMAL	IGE	GTCS
51	MUTHU	43	M		-	+	+		145	43	132	8	4	CVT		PAPILLEDEMA	NOT DONE	CVA-CVT	PSSG
52	SUSHILA	35	F	CKD	-	-	+		113	**221**	130	9	2.2	N		N	NORMAL	UREMIC SEIZURES	GTCS
53	SARAKUTTY	70	F	HTN	-	-	-		102	45	142	8.5	2.5	GLIOTIC CHANGES		HR	ABNORMAL	CVA-ISCHEMIC	SPS
54	MANI	65	M	DM	-	-	-		**54**	56	146	9.2	2.9	N		DR	NOT DONE	HYPOGLYCEMIC SEIZURES	GTCS
55	AROKIASAMY	37	M	PTB	-	+	+	Neck stiffness	95	42	140	8.7	3.1	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
56	PANDARAM	30	M		-	-	+		100	26	136	9.3	3.5	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	GTCS
57	SHEIKH ALI	41	M	HTN	-	-	-		124	52	138	9.5	4.1	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	SPS
58	VAIKATH	28	M		+	-	-	Neck stiffness	106	26	145	8.6	4	N	MEN(B)	N	NOT DONE	BACTERIAL MENINGITIS	GTCS
59	THANGAPERUMA	62	M	DM	-	-	-		**42**	21	135	8.9	3.6	N		DR	NOT DONE	HYPOGLYCEMIC SEIZURES	GTCS
60	MAHADEVI	21	F	ANTENATAL	-	+	+		118	45	139	9.2	4	N		N	NOT DONE	ECLAMPSIA	GTCS
61	ESSAKIAMMAL	28	F		-	+	+		129	38	140	9.5	2.8	CVT		N	NOT DONE	CVA-CVT	GTCS
62	CHARLES	72	M	DM	+	+	-	multiple cranial nerve palsy	152	45	138	8.8	3	CEREBRAL EDEMA			NOT DONE	MUCORMYCOSIS	GTCS
63	PUSHPAM	37	F		-	-	-		110	16	135	9.2	3.5	N		N	ABNORMAL	IGE	GTCS
64	SELVI	24	F		+	-	-	Neck stiffness	149	42	136	8.7	3.6	CEREBRAL EDEMA	MEN(B)	N	NOT DONE	BACTERIAL MENINGITIS	GTCS
65	MADHUBALA	39	F		+	-	-	Neck stiffness	105	26	134	9.2	4.1	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	PAPILLEDEMA	NOT DONE	TB MENINGITIS	GTCS
66	MUKESH	28	M		+	+	-		99	45	142	9.5	3.9	RING ENHANCING LESION		N	NOT DONE	TUBERCULOMA	SPS
67	KAVIPRIYA	37	F	PTB	+	+	-	Neck stiffness	126	39	143	9.3	4.5	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
68	ARUN KUMAR	23	M		-	+	-		135	42	137	8.5	4.6	SOL		N	NOT DONE	TUMOUR	PSSG
69	KABILAN	19	M		+	-	-	-	100	26	144	9.2	3.8	N	MEN(V)	N	NOT DONE	VIRAL MENINGITIS	GTCS
70	RANI	53	F	HTN	-	+	+		131	38	136	8	2.6	HEMORRHAGE		N	NOT DONE	CVA-HEMORRHAGIC	SPS
71	THAVAM PETRAL	45	F	POSTPARTUM	-	+	+		145	45	140	9.5	3.4	N		N	NOT DONE	ECLAMPSIA	SE
72	SUTHAGAR	18	M		-	-	-		104	16	137	9	2.6	N		N	ABNORMAL	IGE	GTCS
73	SENDUR	79	M	PNEUMONIA	-	-	-		154	25	**107**	9.6	2.9	CEREBRAL ATROPHY		N	NOT DONE	HYPONATREMIA	GTCS
74	SENTHIL KUMAR	56	M		-	+	+		140	26	132	9.4	3.1	INFARCT		N	NOT DONE	CVA-ISCHEMIC	GTCS
75	GOPALAKRISHNA	85	M	DM	-	-	-		**39**	48	145	9	3.4	N		N	NOT DONE	HYPOGLYCEMIC	GTCS

76	ESSAKKIAMMAL	32	F		+	+	+	Neck stiffness	178	38	140	8.5	2.9	CEREBRAL EDEMA	MEN(V)	N	NOT DONE	VIRAL MENINGITIS	GTCS
77	SEKHAR	53	M	SHT/DM/CVA	-	+	-		96	54	138	8.8	4.1	INFARCT		N	NOT DONE	CVA-ISCHEMIC	PSSG
78	ANUJA	26	F		+	-	+	Neck stiffness	146	25	133	9	3.7	CEREBRAL EDEMA	MEN(T)	PAPILLED EMA	NOT DONE	TB MENINGITIS	GTCS
79	MEENA	21	F		+	-	+		109	26	130	8.8	8000	INTRACEREBRAL HEMORRHAGE		N	NOT DONE	DENGUE ENCEPHALITIS	SPS
80	VAIRAMUTHU	13	M		-	-	-		135	25	145	8.6	3.6	N		N	NOT DONE	CAMPHOR POISONING	GTCS
81	DIVYA	22	F	ANTENATAL	-	+	+		117	35	142	8.4	2.9	N		N	NORMAL	ECLAMPSIA	GTCS
82	PATTU RAJA	26	M		-	-	-		100	36	138	8.6	4	N		N	ABNORMAL	IGE	GTCS
83	MASANI	67	F	HTN	-	-	-		139	45	144	8.7	4.2	INFARCT		N	NOT DONE	CVA-ISCHEMIC	CPS
84	MADHUMITHA	26	F	ANTENATAL	-	+	+		92	36	136	8.9	4.5	N		N	NORMAL	ECLAMPSIA	GTCS
85	BHAVANI	20	F	POSTPARTUM	-	+	+		133	25	139	9.3	4.6	CVT		PAPILLED EMA	NOT DONE	CVA-CVT	GTCS
86	SELVAKUMAR	14	M		+	+	+	Neck stiffness	95	26	133	9.4	25000	CEREBRAL EDEMA,BILATERAL THALAMIC HYPODENSITY		N	NOT DONE	DENGUE ENCEPHALITIS	GTCS
87	ANTONY RAJ	30	M		-	-	+		120	45	140	8	5	N		N	NORMAL	ALCOHOL WITHDRAWAL	GTCS
88	BALAJI	56	M	HTN	-	+	+		173	54	138	9.5	2.6	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	PSSG
89	MURUGAN	37	M		-	-	-		197	19	135	8.3	3	N		N	ABNORMAL	FAME	GTCS
90	RAJESH	48	M		-	-	-		108	39	140	8.7	2.9	INFARCT		N	NOT DONE	CVA-ISCHEMIC	SPS
91	VEMBU	38	M	CKD	-	-	+		110	**189**	142	9.5	1.6	N		N	ABNORMAL	UREMIC SEIZURES	GTCS
92	PRINCE	28	M		-	-	-		120	42	145	8.5	2.6	N		N	ABNORMAL	IGE	GTCS
93	JEBAKANI	28	F	ANTENATAL	-	+	+		116	36	132	9.2	1.65	NOT DONE		N	NOT DONE	CVA-CVT	SPS
94	ANTONY MICHAE	55	M		-	-	-		163	54	137	9.5	2.6	N		N	ABNORMAL	FAME	GTCS
95	MALLIKA	33	F	POSTPARTUM	-	+	+	multiple cranial nerve palsy	100	35	133	9.3	1.6	CVT		PAPILLED EMA	NOT DONE	CVA-CVT	GTCS
96	SANGEETHA	21	F	ANTENATAL	-	+	+		93	23	136	9.4	2.5	N		N	NOT DONE	ECLAMPSIA	GTCS
97	SELVARAJ	47	M		-	+	+		131	36	142	9.5	3.2	CEREBRAL EDEMA		N	NOT DONE	TUMOUR	CPS
98	SOLAIAPPAN	65	M		+	+	-		126	34	140	8	3.6	SECONDARIES		N	NOT DONE	SECONDARIES BRAIN/CA LUNG	PSSG
99	RAJAN	58	M	DM/CKD	-	-	-		**52**	42	135	8.3	4.5	N		DR	NORMAL	HYPOGLYCEMIC SEIZURES	GTCS
100	CHANDRA	35	M		-	-	-		112	44	132	9.5	4.2	N		N	ABNORMAL	IGE	GTCS
101	KARADI MUTHU	80	M	OLD CVA	-	-	-		156	36	136	9.7	2.6	GLIOTIC CHANGES		N	ABNORMAL	CVA-ISCHEMIC	PSSG
102	SAKTHI GOPAIKA	33	F	POSTPARTUM	-	+	+		113	25	131	9	4.2	N		N	NOT DONE	ECLAMPSIA	GTCS
103	SUBRAMONIAN	33	M	POST THYROID STATUS	-	-	-		143	36	141	**6.4**	3.7	N		N	NOT DONE	HYPOCALCEMIA	GTCS
104	SHANKAR	28	M		+	+	-	Neck stiffness	130	25	148	9.5	4	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	PAPILLED EMA	NOT DONE	TB MENINGITIS	CPS
105	MEENAKUMARI	16	F		+	-	-	Neck stiffness	97	54	142	8.5	4.3	CEREBRAL EDEMA	MEN(V)	PAPILLED EMA	NOT DONE	VIRAL MENINGITIS	GTCS
106	VELLUPANDI	78	M	DM	-	-	-		**550**	23	137	9	4.1	CEREBRAL ATROPHY		N	NOT DONE	HYPERGLYCEMIC	EPC
107	RAMAR	45	M	PTB	-	+	-	Neck stiffness	116	24	134	8.8	4.6	CEREBRAL EDEMA	MEN(T)	N	NOT DONE	TB MENINGITIS	SPS
108	DEIVA	66	M		-	+	+		90	45	136	9.2	2.9	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	GTCS
109	ESSAKKI RAJA	16	M		-	-	-		133	32	140	8.8	3.2	N		N	ABNORMAL	IGE	GTCS
110	SATHEESH KUMA	67	M	HTN	-	-	-		122	26	145	9	4.2	GLIOTIC CHANGES		N	ABNORMAL	CVA-ISCHEMIC	PSSG
111	ASAN IBRAHIM	55	M	PTB	+	+	-		143	36	142	9.5	3.5	RING ENHANCING LESION		N	NOT DONE	TUBERCULOMA	PSSG
112	VEMBU	51	F	DM	-	-	-		**47**	42	146	8.9	4	N		N	NOT DONE	HYPOGLYCEMIC SEIZURES	GTCS
113	MARIAPPAN	37	M		-	-	-		111	68	138	9.2	3.7	N		N	ABNORMAL	FAME	GTCS

114	JEBAKANI	21	F	ANTENATAL	-	+	+		91	56	140	9	4.2	N		N	NOT DONE	ECLAMPSIA	GTCS
115	VEERALAKSHMI	27	F	CKD	-	-	+		128	**208**	139	8.8	4	N		N	NORMAL	UREMIC SEIZURES	GTCS
116	NAMITHA	13	F		+	-	+	Neck stiffness	95	45	144	9.3	3.6	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
117	SEETHALAKSHMI	14	F	T1DM	-	-	-		**56**	62	143	9.5	4.7	N		N	NORMAL	HYPOGLYCEMIC SEIZURES	GTCS
118	NATARAJAN	60	M		-	+	-		154	35	135	8.9	4	INFARCT		N	NOT DONE	CVA-ISCHEMIC	GTCS
119	MATHAN	21	M		-	-	-		175	20	136	9.5	3.8	N		N	ABNORMAL	FAME	GTCS
120	MADHU	47	M		-	+	+	multiple cranial nerve palsy	132	36	139	9.9	4.5	CEREBRAL EDEMA		N	NOT DONE	CVA-CVT	GTCS
121	KAVITHAVENI	24	F		-	-	-		199	23	133	8.6	4.7	N		N	ABNORMAL	IGE	GTCS
122	MUTHU	40	M	HTN	-	+	-		114	45	132	9.2	4.2	INFARCT		HR	NORMAL	CVA-ISCHEMIC	GTCS
123	VIGNESH	13	M		+	+	-	Neck stiffness	93	28	136	9.3	9000	B/L THALAMIC HYPODENSITY		N	NOT DONE	DENGUE ENCEPHALITIS	GTCS
124	CHANDRA	46	F		-	-	-		123	22	140	9	2.9	N		N	ABNORMAL	FAME	GTCS
125	THIRAVIAM	21	M		+	-	-	Neck stiffness	163	36	137	9.5	3	CEREBRAL EDEMA	MEN(V)	PAPILLED EMA	NORMAL	VIRAL MENINGITIS	GTCS
126	GRENA PUSHPAV/	33	F		+	+	-		156	42	143	9.3	13000	B/L THALAMIC HYPODENSITY		N	NORMAL	DENGUE ENCEPHALITIS	GTCS
127	MANTHIRA MOOR	40	M		-	-	-		90	45	142	9.4	4.2	N		N	NORMAL	ALCOHOL WITHDRAWAL	GTCS
128	SUBASH	16	M		-	-	-		143	24	137	8.7	3.6	N		N	ABNORMAL	IGE	GTCS
129	KANNAN	42	M		+	+	+		135	36	133	9	4	SOL		N	NOT DONE	TUMOUR	PSSG
130	MAHENDRAN	43	M	T2DM	-	-	+		**44**	57	140	8.6	3.5	N		N	NORMAL	HYPOGLYCEMIC	SE
131	KARTHIK	28	M		-	-	+		142	24	136	9.2	3.1	N		N	NORMAL	ALCOHOL WITHDRAWAL	GTCS
132	MAHARASI	21	F		+	+	+	Neck stiffness	116	20	130	9.1	30000	N		N	NORMAL	DENGUE ENCEPHALITIS	GTCS
133	STEPHEN	38	M		-	-	-		97	23	134	8.9	2.9	N		N	NORMAL	ALCOHOL WITHDRAWAL	GTCS
134	MUTHULAKSHMI	49	F		-	+	-		112	36	135	8.8	1.8	INFARCT		N	NOT DONE	CVA-ISCHEMIC	GTCS
135	MUNIAMMAL	61	F		-	+	-		98	45	132	9.3	3.5	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	GTCS
136	JAGADEESH	13	M		-	-	+		88	32	135	9.5	4	N		N	NOT DONE	EUCALYPTUS OIL POISONING	GTCS
137	KUMAR	41	M		-	-	+		134	45	140	9	3.2	INFARCT		N	NOT DONE	CVA-ISCHEMIC	GTCS
138	VELSAMY	29	M	HIV	+	+	+	Neck stiffness	132	54	142	9.6	4.2	CEREBRAL EDEMA		N	NOT DONE	CRYPTOCOCCAL MENINGITIS	GTCS
139	CHANDRA	55	F	T2DM/CKD	-	-	-		**40**	78	144	10.2	3.6	N		N	NORMAL	HYPOGLYCEMIC SEIZURES	GTCS
140	MUTHUMARIAPPA	45	F		+	+	+	Neck stiffness	116	26	146	10.3	4.2	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
141	MURUGAMMAL	45	F		-		-		126	54	136	8.8	2.9	SECONDARIES		N	NOT DONE	METASTASIS /CA PAROTID	PSSG
142	ESAKKIAMMAL	17	F		+	+	+		115	26	135	9.2	3.5	CEREBRAL EDEMA	MEN(V)	PAPILLED EMA	NORMAL	VIRAL MENINGITIS	GTCS
143	BHARATHI RAJA	30	M		-	-	+		100	52	140	9.3	4.2	N		N	NOT DONE	OPC POISONING	GTCS
144	SUBRAMONIAN	43	M		-	-	-		83	36	133	8.9	3.7	N		N	NORMAL	ALCOHOL WITHDRAWAL	GTCS
145	MARISELVAM	23	M		+	+	-	Neck stiffness	92	40	145	9.3	3.9	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
146	PONRAJ	46	M		-	-	-		133	25	136	9.5	4.2	INFARCT		N	NORMAL	CVA-ISCHEMIC	GTCS
147	SUFIYA	18	F		+	-	+	Neck stiffness	172	36	140	10.2	2.9	CEREBRAL EDEMA	MEN(B)	PAPILLED EMA	NOT DONE	BACTERIAL MENINGITIS	GTCS
148	JANAKI	58	F		-	-	+		118	37	137	9.6	3.2	HEMORRHAGE		N	NOT DONE	CVA-HEMORRHAGIC	SPS
149	RATHINAM	77	M	HTN/CVA	-	-	-		153	42	132	8.9	4.2	GLIOTIC CHANGES		HR	ABNORMAL	CVA-ISCHEMIC	PSSG
150	SHYAMILI	56	F	DM/HTN	-	-	-		93	36	139	9	4.9	N		MR	NORMAL	HYPERGLCEMIC SEIZURES	GTCS

151	MANIKANDAN	21	M		+	+	-	Neck stiffness	192	56	141	8.7	4.2	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	PAPILLED EMA	NOT DONE	TB MENINGITIS	PSSG
152	PERUMALSAMY	38	M		-	-	-		87	23	143	9.3	3.7	INFARCT		N	NOT DONE	CVA-ISCHEMIC	GTCS
153	STEPHEN RAJ	28	M		+	+	-		173	62	135	9.5	3.6	CALCIFIED GRANULOMA		N	NOT DONE	NCC	SE
154	MANIKANDAN	34	M		-	-	-		210	24	142	8.6	4.1	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	GTCS
155	PETCHIMUTHU	38	M		-	-	+		96	36	146	10	3.8	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	SE
156	ADITYA	18	M		+	-	-	Neck stiffness	113	52	139	9.3	4.1	N	MEN(V)	N	NORMAL	VIRAL MENINGITIS	GTCS
157	NANDINI	16	F		+	-	+		135	23	132	9.5	2.7	CEREBRAL EDEMA	MEN(V)	PAPILLED EMA	ABNORMAL	VIRAL MENINGITIS	GTCS
158	SHANMUGATHAI	70	F	DM/SHT	+	+	+	multiple cranial nerve palsy	107	72	146	10.2	4.2	CEREBRAL EDEMA		N	NOT DONE	MUCORMYCOSIS	GTCS
159	SUMATHI	58	F		-	+	+		110	68	142	9	2.6	SECONDARIES		N	NOT DONE	METASTASIS/CA CERVIX	PSSG
160	JOTHI	41	F		-	-	+		86	**198**	130	9.6	3.4	N		N	NORMAL	UREMIC SEIZURES	GTCS
161	LOGANATH	20	M		-	-	-		77	23	138	9.5	4.2	N		N	ABNORMAL	IGE	GTCS
162	KALEESWARI	28	F		-	-	-		108	18	136	9.7	2.6	N		N	ABNORMAL	FAME	GTCS
163	DURAI	47	M		-	-	-		119	26	145	8.5	3.7	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	GTCS
164	ARAVINDAN	21	M		+	+	-	Neck stiffness	93	35	137	8.8	4	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
165	PUSHPA	32	F	ANTENATAL	-	+	+		100	52	146	9.5	4.2	N		N	NORMAL	ECLAMPSIA	SE
166	ARUMUGAM	19	F		+	+	-	Neck stiffness	123	36	139	9.3	1.6	N	MEN(B)	N	NOT DONE	BACTERIAL MENINGITIS	PSSG
167	VENKADACHALA	48	M		-	-	-		136	26	147	10	1.9	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	GTCS
168	BADHUSHA	21	M		-	-	+		130	52	135	9.5	2.5	N		N	NOT DONE	OPC POISONING	GTCS
169	TAMILARASI	25	F	ANTENATAL	-	+	+		103	62	140	9.6	4.2	NOT DONE		PAPILLED EMA	NOT DONE	CVA-CVT	PSSG
170	PONNU DURAI	55	M	HTN	-	+	-		79	32	132	10	3.7	INFARCT		HR	NOT DONE	CVA-ISCHEMIC	GTCS
171	MANIMEGHALAI	14	F		-	-	-		95	23	136	9.7	4.1	N		N	ABNORMAL	IGE	GTCS
172	SASI	68	M	PTB	+	+	-	Neck stiffness	126	52	149	9.8	3.4	N	MEN(T)	N	NOT DONE	TB MENINGITIS	SPS
173	AYSHA BEEVI	64	F	T2DM	-	-	+		**53**	63	140	10.1	3	N		DR	NORMAL	HYPOGLYCEMIC SEIZURES	GTCS
174	SANYKANNU	55	M	HTN/CVA	-	+	+	Neck stiffness	188	25	145	9.3	2.3	SAH		PAPILLED EMA	NOT DONE	SUB ARACHNOID HEMORRHAGE	GTCS
175	MOHD. ISMAIL	17	M		+	-	+	Neck stiffness	112	40	138	10	3.6	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
176	MANI	38	M		-	+	-		97	23	135	9.5	4.1	SOL		N	NOT DONE	TUMOUR	SPS
177	PERIYASAMY	30	M		-	-	-		122	18	137	10	5.2	N		N	ABNORMAL	IGE	GTCS
178	SARANYA	20	F		-	-	-		111	23	136	9.6	4	N		N	ABNORMAL	IGE	GTCS
179	AYYAKANNU	72	M	OLD CVA	-	-	-		120	38	139	9.8	3.2	GLIOTIC CHANGES		N	ABNORMAL	CVA-ISCHEMIC	PSSG
180	GOPI	41	M		+	+	-	Neck stiffness	78	42	134	10	4.1	N	MEN(V)	N	ABNORMAL	VIRAL MENINGITIS	GTCS
181	BALASUBRAMON	66	M		+	-	+	Neck stiffness	110	26	136	10.5	3	OBSTRUCTIVE HYDROCEPHALUS	MEN(T)	PAPILLED EMA	NOT DONE	TB MENINGITIS	GTCS
182	SARANYA	23	F		+	+	-		201	23	134	9.9	5.1	SOL		N	NOT DONE	TUMOUR	PSSG
183	MADATHY	20	F	ANTENATAL	-	+	+		175	36	136	9.6	4	N		N	NORMAL	ECCLAMPSIA	GTCS
184	SARAVANAN	54	M	HTN	-	+	-		92	69	140	9.3	4.2	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	PSSG

185	MARIAMMAL	27	F	POSTPARTUM	-	+	+		156	36	149	10.5	4.5	CVT		PAPILLEDEMA	NOT DONE	CVA-CVT	PSSG
186	PANEER SELVAN	66	M	DM/CKD	-	-	-		**48**	56	134	10.6	3.7	N		DR	NORMAL	HYPOGLYCEMIC SEIZURES	GTCS
187	SHANMUGHAMM/	68	F	HTN	-	-	-		123	62	135	9	3	HEMORRHAGE		HR	NOT DONE	CVA-HEMORRHAGIC	GTCS
188	MURUGESHWARI	24	F		+	-	-		105	45	140	8.9	4.1	N	MEN(V)	N	ABNORMAL	VIRAL MENINGITIS	PSSG
189	SUBULAKSHMI	14	F		+	+	+	Neck stiffness	85	35	141	9.3	5.1	N	MEN(T)	N	NOT DONE	TB MENINGITIS	GTCS
190	PETER	39	M		-	-	+		99	36	132	8.6	4	N		N	NORMAL	ALCOHOL WITHDRAWAL SEIZURES	GTCS
191	ANNAMMAL	63	F	HTN	-	-	+		118	64	143	9.4	3	INFARCT		HR	NOT DONE	CVA-ISCHEMIC	PSSG
192	SUSHEELA	50	F		+	+	-	Neck stiffness	131	36	135	10	2.6	N	MEN(V)	N	NORMAL	VIRAL MENINGITIS	GTCS
193	MARATHAKAM	28	F		-	-	-		112	21	140	9.2	3.7	N		N	ABNORMAL	IGE	GTCS
194	FATHIMA	19	F		-	-	-		97	20	144	9.5	4.2	N		N	ABNORMAL	FAME	GTCS
195	RADHA	56	F		-	+	-		88	45	138	10	5	INFARCT		N	NOT DONE	CVA-ISCHEMIC	GTCS
196	UDAYAMMAL	73	F	OLD CVA	-	-	-		123	63	139	9.7	2.9	GLIOTIC CHANGES		N	ABNORMAL	CVA-ISCHEMIC	SPS
197	PETCHIMUTHU	26	M		-	-	-		98	18	142	10	3	N		N	ABNORMAL	IGE	GTCS
198	AVUDAYAPPAN	34	M		-	-	-		119	32	139	9.8	4.2	N		N	ABNORMAL	FAME	GTCS
199	ARUNACHALAM	35	M		-	-	-		78	20	135	10.2	3.4	N		N	ABNORMAL	IGE	GTCS
200	PRIYA	32	F	SLE	-	+	+		112	54	134	11	2.9	CVT		N	NOT DONE	CVA- CVT	GTCS
201	VAIDEHI	33	F	POSTPARTUM	+	+	+		116	69	144	9.3	2.8	N		N	NORMAL	ECLAMPSIA	GTCS
202	ESTHER	34	F	CKD/CAD	+	-	+		123	143	140	8.6	1.6	CEREBRAL EDEMA		N	ABNORMAL	POST DIALYSIS	GTCS
203	ANTHONY RAJ	65	M	DM	-	-	+		**50**	54	147	9.3	2.9	CEREBRAL ATROPHY		N	NORMAL	HYPOGLYCEMIC	GTCS
204	PUSHPA RAJ	83	M		-	-	-		94	42	**115**	10.9	3.5	CEREBRAL ATROPHY		N	NORMAL	HYPONATREMIA	GTCS
205	SELVAM	48	M	SHT	-	+	+		98	26	141	9.5	4.6	HEMORRHAGE		N	NOT DONE	CVA -HEMORRHAGIC	PSSG
206	SANKARASELVI	33	F	ANTENATAL	-	+	+		110	62	136	10.2	2.1	N		N	NOT DONE	ECCLAMPSIA	GTCS

age	sex	medical illness	FEVER	HEADACI	VOMITIN
1	1		1	1	0
1	1		1	1	1
6	2	HTN /DM/OLD CVA	1	0	0
4	1	HTN/DM	0	0	0
1	2		1	1	0
1	2		0	0	0
2	2	CKD	0	0	1
2	2	0	0	1	0
6	2	CA LUNG	0	1	0
3	1		0	0	0
2	2	0	0	1	1
1	2		0	0	1
4	1		0	0	1
3	2	1	0	1	0
7	1	DM	0	0	0
6	2	DM	0	0	0
4	2		0	0	0
2	1		0	1	0
6	1	HTN	0	0	0
6	1	DM	0	0	1
5	1	HTN	0	1	0
2	1		1	1	1
1	2		0	0	0
5	1		0	0	0
1	1		0	0	0
5	2		0	0	0
4	2		0	0	0
3	1		0	0	0
2	1		0	0	0
1	2		1	1	0
4	2		0	0	0
3	2	1	0	1	1
4	2		0	0	0
7	1		0	0	0
1	2	HTN	0	0	0
1	1	T1DM	0	0	0
3	1		0	1	1
7	1		0	0	0
2	2	1	0	1	1
3	2		0	0	0
5	1		0	0	0
2	2		0	0	0
2	2		0	0	0
3	1		0	1	0
1	1		1	0	0
6	2		0	1	1
4	1		0	0	0
3	2		0	0	0
5	1		0	1	0

2	2		0	0	0
4	1		0	1	1
3	2	CKD	0	0	1
6	2		0	0	0
6	1	DM	0	0	0
3	1		0	1	1
2	1		0	0	1
4	1		0	0	0
2	1		1	0	0
6	1	DM	0	0	0
2	2	1	0	1	1
2	2	0	0	1	1
7	1	DM	1	1	0
3	2		0	0	0
2	2		1	0	0
3	2		1	0	0
2	1		1	1	0
3	2		1	1	0
2	1		0	1	0
1	1		1	0	0
5	2		0	1	1
4	2	0	0	1	1
1	1		0	0	0
7	1	PNEUMONIA	0	0	0
5	1		0	1	1
8	1	DIABETES	0	0	0
3	2		1	1	1
5	1	SHT/DM/CVA	0	1	0
2	2		1	0	1
2	2		1	0	1
1	1		0	0	0
2	2	1	0	1	1
2	1		0	0	0
6	2		0	0	0
2	2	0	0	1	1
1	2	1	0	1	1
1	1		1	1	1
2	1		0	0	1
5	1		0	1	1
3	1		0	0	0
4	1		0	0	0
3	1		0	0	1
2	1		0	0	0
2	2	1	0	1	1
5	1		0	0	0
3	2	0	0	1	1
2	2	1	0	1	1
4	1		0	1	1
6	1		1	1	0
5	1		0	0	0

3	1		0	0	0
7	1		0	0	0
3	2	1	0	1	1
3	1	POST THYROID STATU	0	0	0
2	1		1	1	0
1	2		1	0	0
7	1	DM	0	0	0
4	1	TB	0	1	0
6	1		0	1	1
1	1		0	0	0
6	1		0	0	0
5	1		1	1	0
5	2		0	0	0
3	1		0	0	0
2	2	1	0	1	1
2	2		0	0	1
1	2		1	0	1
1	2	T1DM	0	0	0
5	1		0	1	0
2	1		0	0	0
4	1		0	1	1
2	2		0	0	0
4	1	SHT	0	1	0
1	1		1	1	0
4	2		0	0	0
2	1		1	0	0
3	2		1	1	0
3	1		0	0	0
1	1		0	0	0
4	1		1	1	1
4	1	T2DM	0	0	1
2	1		0	0	1
2	2		1	1	1
3	1		0	0	0
4	2		0	1	0
6	2		0	1	0
1	1		0	0	1
4	1		0	0	1
2	1	HIV	1	1	1
5	2	T2DM/CKD	0	0	0
4	2		1	1	1
4	2		0		0
1	2		1	1	1
2	1		0	0	1
4	1		0	0	0
2	1		1	1	0
4	1		0	0	0
1	2		1	0	1
5	2		0	0	1
7	1		0	0	0

5	2	DM/SHT	0	0	0
2	1		1	1	0
3	1		0	0	0
2	1		1	1	0
3	1		0	0	0
3	1		0	0	1
1	1		1	0	0
1	2		1	0	1
6	2	DM/SHT	1	1	1
5	2		0	1	1
4	2		0	0	1
1	1		0	0	0
2	2		0	0	0
4	1		0	0	0
2	1		1	1	0
3	2	1	0	1	1
1	2		1	1	0
4	1		0	0	0
2	1		0	0	1
2	2	1	0	1	1
5	1		0	1	0
1	2		0	0	0
3	1		1	1	0
6	2	T2DM	0	0	1
5	1	HTN/CVA	0	1	1
1	1		1	0	1
3	1		0	1	0
2	1		0	0	0
1	2		0	0	0
7	1		0	0	0
6	1		1	1	0
5	1		1	0	1
2	2		1	1	0
1	2	1	0	1	1
5	1	HTN	0	1	0
2	2	0	0	1	1
6	1	DM/CKD	0	0	0
6	2	HTN	0	0	0
2	2		1	0	0
1	2		1	1	1
3	1		0	0	1
6	2		0	0	1
4	2		1	1	0
2	2		0	0	0
1	2		0	0	0
5	2		0	1	0
7	2		0	0	0
2	1		0	0	0
3	1		0	0	0
3	1		0	0	0

3	2	SLE	0	1	1
3	2	0	1	1	1
3	2	CKD/CAD	1	0	1
6	1	DM	0	0	1
8	1		0	0	0
4	1	SHT	0	1	1
3	2	1	0	1	1

NEURO E	RBS	urea	Na	Ca	platelet	CT brain	CSF
0	0	0	0	0	0	1	0
0	0	0	0	0	0	1	1
1	0	0	0	0	0	1	
	1	0	0	0	0	0	
0	0	0	0	0	0	1	2
	0	0	0	0	0	0	
	0	**1**	0	0	0	0	
	0	0	0	0	0	1	
1	0	0	0	0	0	1	
	0	0	0	0	0	0	
paraparesis	0	0	0	0	0	1	
	0	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
DEMENTIA	0	0	0	0	0	1	
	1	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
1	0	0	0	0	0	1	
	0	0	**1**	0	0	0	
0	0	0	0	0	0	1	
0	0	0	0	0	0	1	0
	0	0	0	0	0	0	
1	0	0	0	0	0	1	
	0	0	0	0	0	0	
	2	0	0	0	0	0	
1	0	0	0	0	0	1	
	0	0	0	0	0	1	
	0	0	0	0	0	0	
0	0	0	0	0	0	1	0
	0	0	0	0	0	0	
	0	0	0	0	0	0	
1	0	0	0	0	0	1	
1	0	0	0	0	0	1	
HEMIPLEGIA	0	0	0	9	0	1	
	1	0	0	0	0	0	
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1	0	0	0	0	0	1	
	0	0	0	0	0	0	
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1	0	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
0	0	0	0	0	0	0	1
0	0	0	0	0	0	1	
	0	0	0	0	0	1	
	0	0	0	**1**	0	0	
	0	0	0	0	0	1	

	0	0	0	0	0	0	
	0	0	0	0	0	1	
	0	**1**	0	9	0	0	
	0	0	0	0	0	1	
	1	0	0	0	0	0	
0	0	0	0	0	0	1	0
	0	0	0	0	0	0	
	0	0	0	0	0	1	
0	0	0	0	0	0	0	2
	1	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
	0	0	0	0	0	1	
	0	0	0	0	0	0	
0	0	0	0	0	0	1	2
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
-	0	0	0	0	0	0	1
	0	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	**1**	0	0	0	
	0	0	0	0	0	1	
	1	0	0	0	0	0	
0	0	0	0	0	0	0	1
	0	0	0	0	0	1	
0	0	0	0	0	0	1	0
	0	0	0	0	1	1	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
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	0	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
	0	**1**	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	99	
	0	0	0	0	0	0	
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	0	0	0	0	0	1	
	0	0	0	0	0	1	
	1	0	0	0	0	0	

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	0	0	0	0	0	0	
	0	0	0	1	0	0	
0	0	0	0	0	0	1	0
0	0	0	0	0	0	1	1
	2	0	0	0	0	0	
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
	0	0	0	0	0	1	
	1	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	**1**	0	0	0	0	
0	0	0	0	0	0	1	0
	1	0	0	0	0	0	
	0	0	0	0	0	1	
	0	0	0	0	0	0	
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	0	0	0	0	0	0	
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0	0	0	0	0	0	0	1
	0	0	0	0	1	1	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	1	
	1	0	0	0	0	0	
	0	0	0	0	0	0	
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	0	0	0	0	0	1	
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	1	0	0	0	0	0	
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
	0	0	0	0	0	1	1
	0	0	0	0	0	0	
	0	0	0	0	0	0	
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
0	0	0	0	0	0	1	2
	0	0	0	0	0	1	
	0	0	0	0	0	1	

	0	0	0	0	0	0	
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
	0	0	0	0	0	1	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
0	0	0	0	0	0	0	1
	0	0	0	0	0	1	1
multiple cranial nerve palsy	0	0	0	0	0	1	
	0	0	0	0	0	1	
	0	**1**	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	0	
0	0	0	0	0	0	0	0
	0	0	0	0	0	0	
0	0	0	0	0	0	1	2
	0	0	0	0	0	0	
	0	0	0	0	0	0	
	0	0	0	0	0	99	
	0	0	0	0	0	1	
	0	0	0	0	0	0	
0	0	0	0	0	0	1	0
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0	0	0	0	0	0	1	
0	0	0	0	0	0	1	0
	0	0	0	0	0	1	
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	0	0	0	0	0	0	
	0	0	0	0	0	1	
0	0	0	0	0	0	1	1
0	0	0	0	0	0	1	0
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	0	0	0	0	0	0	
	0	0	0	0	0	1	
	0	0	0	0	0	1	
	1	0	0	0	0	0	
	0	0	0	0	0	1	
	0	0	0	0	0	0	1
0	0	0	0	0	0	1	0
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	0	0	0	0	0	1	
0	0	0	0	0	0	1	1
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	0	0	0	0	0	0	
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0	0	0	0	0	1
0	0	0	0	0	0
0	0	0	0	0	0
1	0	0	0	0	0
0	0	**1**	0	0	0
0	0	0	0	0	1
0	0	0	0	0	0

FUNDUS	EEG fir	MRI	dia	DIAGNOSIS	infections	CVA	metaboli	tumor
0			1	1	0			
0			1	1	4			
			2	CVA-ischemic		0		
			3	HYPOGLYCEMIC			0	
	BIlater		1	BACTERIAL MENINGITIS	2			
	N		6	6				
			3	UREMIC SEIZURES			4	
1			2	CVA-CVT		2		
			4	SECONDARIES BRAIN/CA				0
			7	ALCOHOL WITHDRAWAI				
1	BILATE		2	CVA-CVT		2		
	BILATE		4	TUMOUR				1
			5	OPC POISONING				
			8	ECLAMPSIA				
			2	CVA-ISCHEMIC		0		
	NORM		3	HYPOGLYCEMIC SEIZURI			0	
	BILATE		6	IGE				
	SPIKE &		4	TUMOUR				1
	NORM		2	CVA-HEMORRHAGIC		1		
	BILATE		3	HYPONATREMIA			3	
	NORM		2	SUB ARACHNOID HEMO		3		
	NORM		1	TB MENINGITIS	0			
	NORM		9	FAME				
	NORM		2	CVA-ISCHEMIC		0		
	R FROI		6	IGE				
	NORM		3	HYPERGLYCEMIC SEIZUR			1	
	NORM		1	TUBERCULOMA	6			
			1	NEUROCYSTICERCOSIS	5			
	BILATE		7	ALCOHOL WITHDRAWAI				
			1	TB MENINGITIS	0			
	NORM		6	IGE				
	NORM		8	ECLAMPSIA				
	BILATE		2	CVA-HEMORRHAGIC		1		
	NORM		2	CVA-ISCHEMIC		0		
			2	CVA-HEMORRHAGIC		1		
	BILATE		3	HYPOGLYCEMIC SEIZURI			0	
1	BILATE		2	CVA-CVT		2		
	BILATE		2	CVA-ISCHEMIC		0		
	BILATE		8	ECLAMPSIA				
	BILATE		5	OPC POISONING				
	R FROI		2	CVA-ISCHEMIC		0		
	NORM		9	FAME				
	BILATE		6	IGE				
			2	SDH		4		
	BILATE		1	VIRAL MENINGITIS	4			
	BILATE		2	SUB ARACHNOID HEMO		3		
	R FROI		2	CVA-ISCHEMIC		0		
	NORM		3	HYPOCALCEMIA			2	
	OCC SI		2	CVA-ISCHEMIC		0		

	BILATE	6	IGE				
1	BILATE	2	CVA-CVT		2		
		3	UREMIC SEIZURES			4	
	BILATE	2	CVA-ISCHEMIC		0		
	NORM	3	HYPOGLYCEMIC SEIZURI			0	
		1	TB MENINGITIS	0			
	NORM	7	ALCOHOL WITHDRAWAI				
	SPIKE &	2	CVA-HEMORRHAGIC		1		
		1	BACTERIAL MENINGITIS	2			
		3	HYPOGLYCEMIC SEIZURI			0	
		8	ECLAMPSIA				
0	NORM	2	CVA-CVT		2		
	NORM	1	MUCORMYCOSIS	1			
	BILATE	6	IGE				
		1	BACTERIAL MENINGITIS	2			
		1	TB MENINGITIS	0			
		1	TUBERCULOMA	6			
		1	TB MENINGITIS	0			
		4	TUMOUR				1
		1	VIRAL MENINGITIS	4			
0		2	CVA-HEMORRHAGIC		1		
	N	8	ECLAMPSIA				
	BILATE	6	IGE				
		3	HYPONATREMIA			3	
		2	CVA-ISCHEMIC		0		
	N	3	HYPOGLYCEMIC			0	
		1	VIRAL MENINGITIS	4			
		2	CVA-ISCHEMIC		0		
		1	TB MENINGITIS	0			
		1	DENGUE ENCEPHALITIS	3			
		5	CAMPHOR POISONING				
	BEA	8	ECLAMPSIA				
	N	6	IGE				
		2	CVA-ISCHEMIC		0		
	N	8	ECLAMPSIA				
1	BEA	2	CVA-CVT		2		
		1	DENGUE ENCEPHALITIS	3			
	N	7	ALCOHOL WITHDRAWAI				
		2	CVA-HEMORRHAGIC		1		
	N	9	FAME				
		2	CVA-ISCHEMIC		0		
		3	UREMIC SEIZURES			4	
	N	6	IGE				
0		2	CVA-CVT		2		
		9	FAME				
1		2	CVA-CVT		2		
		8	ECLAMPSIA				
		4	TUMOUR				1
		4	SECONDARIES BRAIN/CA				0
		3	HYPOGLYCEMIC SEIZURI			0	

	N	6	IGE					
		2	CVA-ISCHEMIC		0			
		8	ECLAMPSIA					
	N	3	HYPOCALCEMIA			2		
	R TEM	1	TB MENINGITIS	0				
	BL SPII	1	VIRAL MENINGITIS	4				
	N	3	HYPERGLYCEMIC			1		
	N	1	TB MENINGITIS	0				
	PAROX	2	CVA-HEMORRHAGIC		1			
	BL FRC	6	IGE					
	BL SPII	2	CVA-ISCHEMIC		0			
	BL FRC	1	TUBERCULOMA	6				
		3	HYPOGLYCEMIC SEIZURI			0		
	BL SYN	9	FAME					
		8	ECLAMPSIA					
		3	UREMIC SEIZURES			4		
		1	TB MENINGITIS	0				
		3	HYPOGLYCEMIC SEIZURI			0		
	N	2	CVA-ISCHEMIC		0			
		9	FAME					
0		2	CVA-CVT		2			
	N	6	IGE					
	PAROX	2	CVA-ISCHEMIC		0			
		1	DENGUE ENCEPHALITIS	3				
	BL FRC	9	FAME					
		1	VIRAL MENINGITIS	4				
		1	DENGUE ENCEPHALITIS	3				
		7	ALCOHOL WITHDRAWAI					
	N	6	IGE					
		4	TUMOUR				1	
		3	HYPOGLYCEMIC			0		
	N	7	ALCOHOL WITHDRAWAI					
	N	1	DENGUE ENCEPHALITIS	3				
		7	ALCOHOL WITHDRAWAI					
		2	CVA-ISCHEMIC		0			
		2	CVA-HEMORRHAGIC		1			
		5	EUCALYPTUS OIL POISOI					
		2	CVA-ISCHEMIC		0			
		1	CRYPTOCOCCAL MENIN(	1				
		3	HYPOGLYCEMIC SEIZURI			0		
		1	TB MENINGITIS	0				
		4	METASTASIS /CA PAROT				0	
		1	VIRAL MENINGITIS	4				
		5	OPC POISONING					
	N	7	ALCOHOL WITHDRAWAI					
	N	1	TB MENINGITIS	0				
	N	2	CVA-ISCHEMIC		0			
	BEA	1	BACTERIAL MENINGITIS	2				
	B/L SH	2	CVA-HEMORRHAGIC		1			
	ABNOI	2	CVA-ISCHEMIC		0			

		3	HYPERGLCEMIC SEIZURE			1		
		1	TB MENINGITIS	0				
		2	CVA-ISCHEMIC		0			
		1	NCC	5				
	N	7	ALCOHOL WITHDRAWAI					
	N	7	ALCOHOL WITHDRAWAI					
	N	1	VIRAL MENINGITIS	4				
		1	VIRAL MENINGITIS	4				
		1	MUCORMYCOSIS	1				
		4	METASTASIS/CA CERVIX					0
		3	UREMIC SEIZURES			4		
	N	6	IGE					
		9	FAME					
		7	ALCOHOL WITHDRAWAI					
		1	TB MENINGITIS	0				
		8	ECLAMPSIA					
		1	BACTERIAL MENINGITIS	2				
		7	ALCOHOL WITHDRAWAI					
		5	OPC POISONING					
1		2	CVA-CVT		2			
		2	CVA-ISCHEMIC		0			
	N	6	IGE					
		1	TB MENINGITIS	0				
		3	HYPOGLYCEMIC SEIZURI			0		
		2	SUB ARACHNOID HEMO		3			
		1	TB MENINGITIS	0				
		4	TUMOUR					1
	N	6	IGE					
	N	6	IGE					
		2	CVA-ISCHEMIC		0			
		1	VIRAL MENINGITIS	4				
		1	TB MENINGITIS	0				
		4	TUMOUR					1
		8	ECCLAMPSIA					
		2	CVA-HEMORRHAGIC		1			
1		2	CVA-CVT		2			
		3	HYPOGLYCEMIC SEIZURI			0		
		2	CVA-HEMORRHAGIC		1			
		1	VIRAL MENINGITIS	4				
		1	TB MENINGITIS	0				
		7	ALCOHOL WITHDRAWAI					
		2	CVA-ISCHEMIC		0			
		1	VIRAL MENINGITIS	4				
	N	6	IGE					
		9	FAME					
		2	CVA-ISCHEMIC		0			
		2	CVA-ISCHEMIC		0			
	N	6	IGE					
		9	FAME					
	N	6	IGE					

2	CVA- CVT	2	
8	ECLAMPSIA		
0	POST DIALYSIS		
3	HYPOGLYCEMIC		0
3	HYPONATREMIA		3
2	CVA -HEMORRHAGIC		
8	ECCLAMPSIA		

poisonin	pregnancy related
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AGE	sex	fever	headache	vomiting	neuro	rbs	urea	sodium	calcium
13-20=1	m=1	yes=1	yes=1	yes=1		normal=0	normal=0	normal=0	normal=0
21-30=2	f=2	no=0	no=0	no=0		hypo=1	high=1	hypo=1	hypo=1
31-40=3						hyper=2			
41-50=4									
51-60=5									
61-70=6									
71-80=7									
>80=8									

platelet	CSF	ct brain	fundus	eeg	diag
normal=0	T=0	normal=0	normal=0	normal=0	cns infection=1
hypo=1	V=1	abnormal=1	abnormal=1	abnormal=1	cva=2
	B=2	NOT DONE=99			metabolic=3
					tumor=4
					poisoning=5
					idiopathic=6
					alcohol withdrawal=7
					ecclampsia=8
					fame=9
					post dialysis=0

cns infection	CVA	metabolic	tumor	poisoning
tbm=0	infarct=o	Hypoglycemia=0	secondaries=0	OPC=0
fungal=1	hemorrhage=1	hyperglycemia=1	tumor=1	others=1
bacterial=2	CVT=2	hypocalcemia=2		
dengue=3	SAH=3	hyponatremia=3		
other viral=4	SDH=4	uremic=4		
ncc=5				
tuberculoma=6				

FUNDUS	PREGNANCY RELATED=1	ANTENATAL=1
N=0	UNRELATED=0	POSTPARTUM=0
PAPILLEDEMA=1		